CARDIAC ARRHYTHMIAS

UNIFORM WITH THIS VOLUME

THE BLOOD SUPPLY TO THE HEART IN ITS ANATOMICAL AND CLINICAL ASPECTS

BY
LOUIS GROSS, M.D., C.M.
New York

PAUL B. HOEBER, INC., *Publishers*
SEVENTY-SIX FIFTH AVE. · NEW YORK

CARDIAC ARRHYTHMIAS

CLINICAL FEATURES & MECHANISM OF THE IRREGULAR HEART

BY

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INTRODUCTION BY

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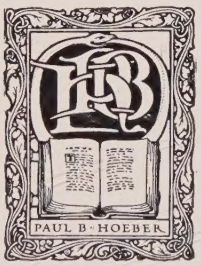
WITH EIGHTY ILLUSTRATIONS AND FIVE TABLES



PAUL B · HOEBER · INC

NEW YORK · MCMXXVIII

PUBLISHED MARCH, 1928

Printed in the United States of America

TO THE
MEMORY OF MY DEAR MOTHER TO WHOM
THE PLEASURE OF SEEING THIS BOOK
COMPLETED HAS BEEN DENIED BY THE INEVITABLE
DESTINY OF MAN, AND TO MY FRIEND AND
TEACHER, DR. JOHN C. CARDWELL,
WHO WAS THE FIRST TO KINDLE
MY INTEREST IN PHYSIOL-
OGY, THIS BOOK IS AF-
FECTIONATELY
DEDICATED

INTRODUCTION

The impulse to prepare this volume came as a result of an exhibit made by Dr. Roth at the meeting of the American Medical Association in May, 1925. When Dr. Alexander Lambert, Dr. S. Calvin Smith and the writer were organizing an exhibit in cardiology for that meeting we were strongly advised to invite Dr. Roth to show a collection of graphic representations of the mechanism and the clinical features of the arrhythmias, which he had for some time been employing in his teaching. His exhibit was widely admired and its importance for purposes of instruction so much appreciated, that Dr. Roth was urged to publish his diagrams in book form. This he has done in this volume with sufficient text to describe the normal anatomy and physiology of the heart, and the mechanism and clinical characteristics of the various types of arrhythmia.

The teaching value of the graphs, which include not only the cardiac mechanism involved, but also the correlated heart sounds and arterial and venous pulses, is evident. The diagrammatic representations of the mechanism of auricular fibrillation and auricular flutter are original and particularly striking. Dr. Roth has succeeded in writing a simple, clear text. I know of no other book which makes the mechanism and significance of the arrhythmias more easily understandable by the uninitiated. Particularly praiseworthy is the attention paid to the clinical side of the subject and the emphasis laid upon recognizing as much as possible by the unaided senses. The reason is given in the following impressive way: " . . . since it is the man and not the instrument who ought to be the final

judge, the potentialities of our special senses as simple, useful and readily available means in clinical medicine ought to be developed and utilized."

This volume should be particularly valuable to the general clinician who has so often neglected opportunities of acquainting himself with the subject of the irregularities of the heart because of his fear of the subject being too complex for his understanding. I feel confident that it will receive the cordial reception from the profession that it merits.

E. Libman.

New York, N. Y.
February, 1928.

PREFACE

The generic phrase "irregular heart" has until recently included a motley group of cardiac arrhythmias, which the clinician, because of the lack of comprehensive criteria, could not differentiate into its component types. Even elaborate texts on physical diagnosis treated the subject with but scant comment. Clinical inquiry into cardiac disorders has been directed largely toward the investigation of valvular defects and their concomitant features. Altered hemodynamics, as manifested by murmurs, changes in area of cardiac dullness and changes in type of arterial pulse beat, seem to have attracted most attention.

The clinical features of the various types of cardiac irregularity have been thus far generally disregarded even by those most closely in touch with them, the clinicians. It has been left to the physiologist and to those physicians especially interested in cardiovascular disorders, who have had access to instruments of precision, such as the polygraph and the electrocardiograph, to decipher the mechanism of the cardiac arrhythmias. Fortunately, however, not only have they revealed to us these mechanisms, but they have also given us numerous valuable clues to clinical signs. The clinician may now avail himself of the existing knowledge of these mechanisms and, by observing and correlating the available clinical signs, arrive at helpful clinical criteria. By aid of such criteria the various types of irregular heart action may become clinically comprehensible in a large number of instances with nearly as much facility as are the valvular defects by aid of their criteria, murmurs, etc.

It is the aim of this volume to present in simple diagrammatic form the mechanism of the various types of cardiac irregularity together with their concomitant extracardiac manifestations (clinical signs), such as, electrical manifestations, heart sounds and the arterial and venous pulses. All diagrams are drawn to scale in order that the various components of the extracardiac manifestations of the heart beat may be correlated with one another and with the corresponding phases of the intrinsic cardiac mechanism, in any given arrhythmia. The attempt is also made to simplify by aid of diagrams, the understanding of that disturbance in the intrinsic cardiac mechanism known as "Circus Movement" which is accepted today as the underlying disturbance of rhythm in clinical auricular flutter and fibrillation.

The effort is made to reduce text matter to a minimum. In its place, liberal use is made of diagrams and graphs to depict the interrelation of the various clinical phenomena in the normal as well as in the irregular heart beat.

The first part of this book is devoted to a review of such anatomical and physiological facts as may seem essential to the understanding of the mechanism of the normal heart beat. Following this, it presents a brief study of the electrocardiogram, heart sounds and the arterial and venous pulses, discussing their mode of genesis, characteristic clinical features and the usual instrumental and bedside methods employed in their study. The first part, dealing with normal phenomena, serves as a preparation for a more ready appreciation of those alterations in heart action which may constitute some of the arrhythmias.

The second part deals with the arrhythmias exclusively. These are classified under five general groups, divided under

component sub-groups. Each sub-group is described with the aid of diagrams, depicting the disturbance in cardiac mechanism and the characteristic alterations in all the accompanying clinical phenomena, electrical changes, heart sounds and arterial and venous pulses. *Clinical signs and symptoms are stressed throughout the volume.* At the end of every chapter the clinical signs and symptoms of each component disorder of the given group are tabulated. Such tables, presenting the distinguishing clinical features of each group in a condensed form, tend to serve as clinical criteria for differential diagnosis.

It is hoped that this book will accomplish a useful purpose in awakening a wider interest in the *bedside study* of the irregular heart beat. It may serve also to introduce and simplify the study of graphic methods. These, because of the highly specialized instruments employed and because of a new nomenclature which has arisen, seem unduly complicated and therefore comparatively unattractive to the average practitioner and student.

The condensation of a broad subject into a volume of reasonable size is obviously difficult and is always attended with the danger of omissions; however, in order to make it more readily comprehensible, and particularly in order to bring it within reach of those who have neither time nor inclination to make a special study of circulatory phenomena, condensation of the subject becomes a prime requisite.

There is no scarcity of large volumes on disorders of the cardiac rhythm. Pioneers, such as Mackenzie, Wenckebach, Lewis and others, have presented the subject in great detail. Such presentations, however, having been generally but a part of otherwise extensive and detailed polygraphic and electrocardiographic studies, are apparently too intricate for

the uninitiated. In contrast to these works, the present volume aims to offer the elements of graphic studies in the arrhythmias and to emphasize their clinical features.

In preparing my manuscript, I have had the good fortune to receive the friendly, constructive criticism of the following men, to whom I am truly grateful: Prof. H. B. Williams for his review of the chapter on electrocardiography; Dr. B. S. Oppenheimer for his valuable criticism on the chapter on cardiac anatomy and physiology, and on auricular flutter and fibrillation. Particularly am I indebted to Drs. Alexander Lambert, Emanuel Libman and Marcus A. Rothschild, for their friendly encouragement, aid and criticism throughout the preparation of this volume.

I would seem ungrateful indeed, if I failed to express my sincere thanks to my publisher, Mr. Paul B. Hoeber and his staff for their untiring efforts and excellent cooperation.

IRVING R. ROTH.

NEW YORK, N. Y.
February, 1928.

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CARDIAC ARRHYTHMIAS

Introduction to Part One

THE proper understanding of the mechanism of the heart beat presupposes an understanding of some of the fundamental anatomical and physiological facts about the heart.

Anatomically, the parenchyma of the heart consists essentially of muscle. The heart muscle composing the walls of the chambers proper consists of groups of striated muscle cells joined to form a syncytium. These groups of muscle cells, layer upon layer, intertwining and encircling the chambers, constitute the real functional contractile elements of the heart (Fig. 1).

The Specialized Muscle Tissue of the Heart

Besides these, however, the walls of the heart contain groups, nests as it were, of a specialized neuromuscular tissue, collectively constituting the "Keith-Tawara-His system." At times they are also referred to as the "specific system" or merely as the "specialized system." (Fig. 2.) In the text that follows, these names will be used interchangeably. These "nests" of neuromuscular tissue resemble more nearly embryonic muscle tissue and differ in many respects from the ordinary cardiac muscle, both morphologically and physiologically. The uppermost of these groups of specialized cells is situated in the wall of the right auricle at its junction with the superior vena cava and is known as the *sino-auricular node*. Another, the *auriculo-ventricular node*, is situated in the posterior-inferior portion of the interauricular septum. From this point on, the

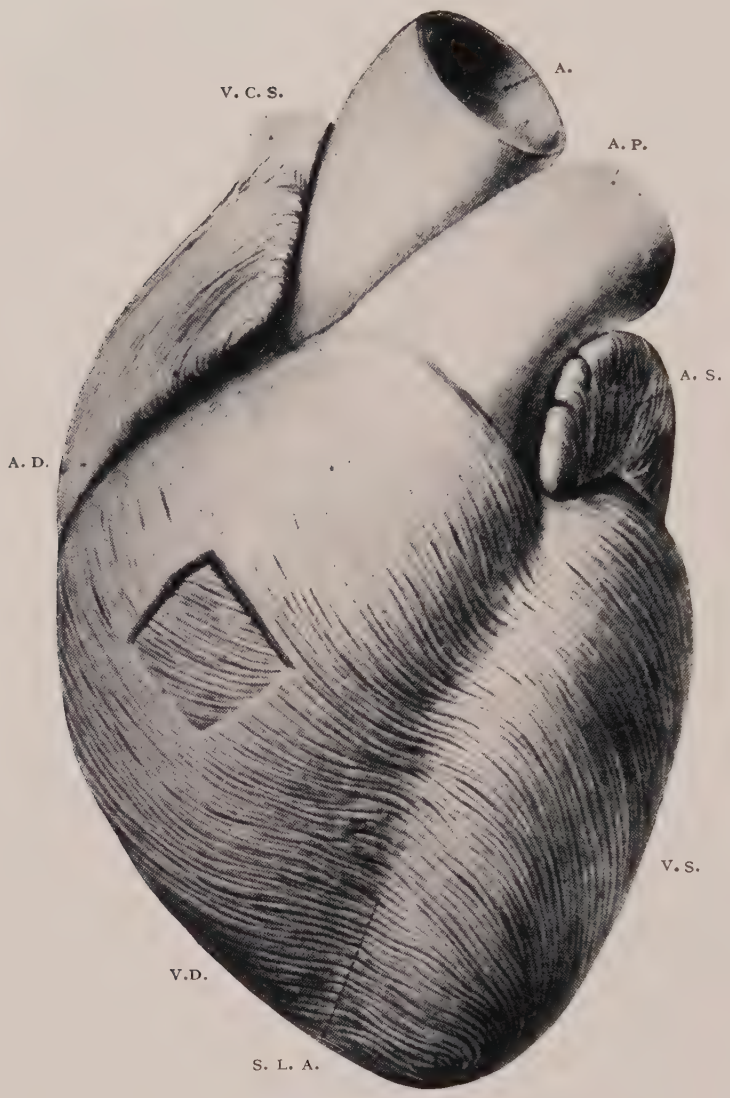

FIG. 1.—The general arrangement of the heart muscle composing the walls of the cardiac chambers. These constitute the true contractile elements of the heart. A. aorta; A.D. atrium dextrum; A.P. arteria pulmonalis; A.S. auricula sinistra; S.L.A. sulcus longitudinalis anterior; V.C.S. vena cava superior; V.D. ventriculus dexter; V.S. ventriculus sinister. (*From Sobotta.*)

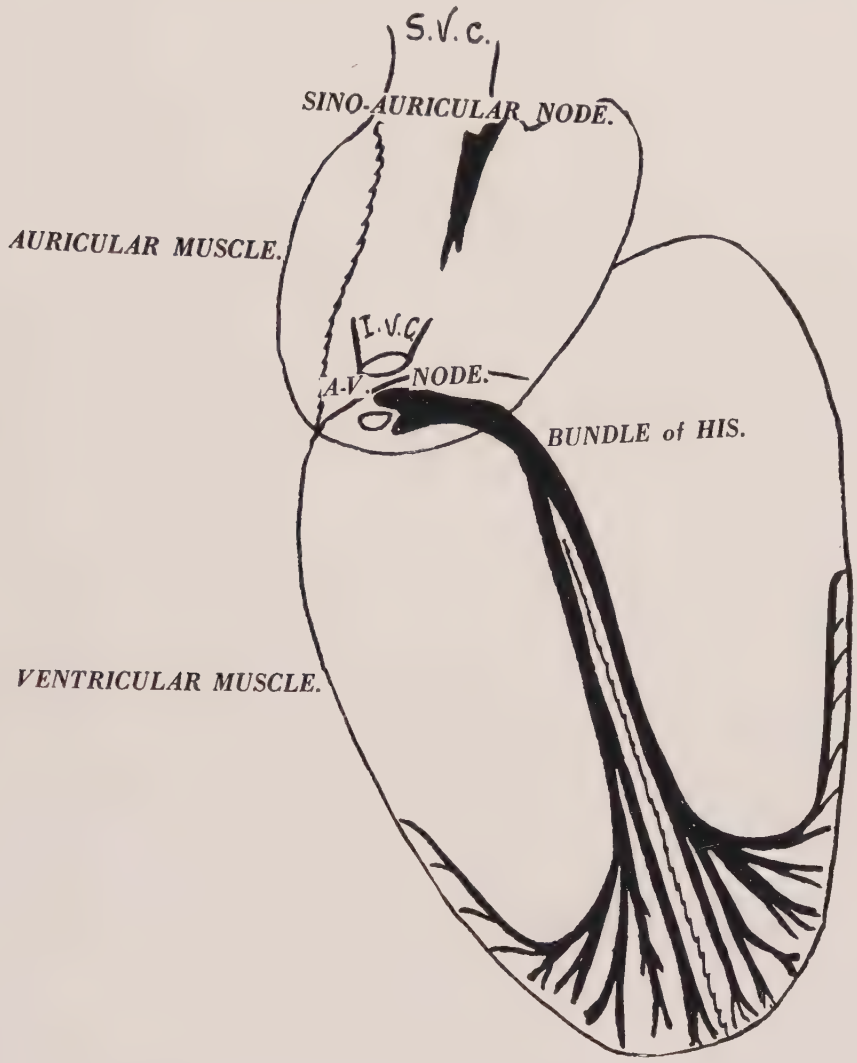

FIG. 2.—Schematic drawing of a transparent heart, showing auricles, ventricles, superior vena cava (S.V.C.) and inferior vena cava (I.V.C.). The heavily shaded areas represent the "specific system." (Keith-Tawara-His system.)

"system" is continuous as a special bundle, the *bundle of His*, joining auricles and ventricles. Within the ventricles this bundle subdivides into *right and left branches*, one for each ventricle. Coursing beneath the endocardium, on either side of the interventricular septum, each of these main branches further subdivides and their *terminal arborizations* are extensively distributed throughout the subendocardial layers of both ventricles, ultimately coming into intimate contact with the ordinary heart muscle cells of these chambers.

Significance of the Specialized Neuromuscular Tissue in the Arrhythmias

A clear understanding of the anatomy and physiology of the specific system is absolutely essential to a clear understanding of the cardiac mechanism and consequently of the mechanism of the arrhythmias. The rhythmic production of the cardiac stimulus and its sequential propagation along its usual course *depends upon the integrity of the specific system.*

The arrhythmias have their genesis in the anatomical or functional disturbance of one or more of the subdivisions of this system. Although the ordinary heart muscle may be extensively diseased or in part destroyed and replaced by fibrous tissue, the heart may still beat regularly, and in fact it generally does; however, as soon as the pathological process extends to and involves the specialized tissues, arrhythmias usually supervene. It is evident, therefore, that a knowledge of the anatomical and physiological peculiarities of the specific system is a fundamental prerequisite to the understanding of the cardiac arrhythmias.

Chapter I

The Specific System (Keith-Tawara-His System)

1. Anatomical Subdivisions

THE specific neuromuscular system may be subdivided anatomically into these groups:

1. The Sino-Auricular Node (S-A Node) or Keith-Flack Node

2. The Auriculo-Ventricular Node (A-V Node) or Node of Tawara

3. The Bundle Proper or His' Bundle

4. The Right and Left Divisions of the Bundle (Bundle Branches)

5. The Terminal Arborizations

6. The Network of Purkinje.

1. THE SINO-AURICULAR NODE

This structure is situated at the junction of the superior vena cava and the right auricle (Fig. 3). It extends caudad, lying within the substance of a shallow groove, the sulcus terminalis, which corresponds to the embryonic union of the right auricle and its appendage. The node is about 2 cm. long and about 2 mm. thick. It is embedded in connective tissue and has a rich nerve and blood supply.

The muscle fibers of this node are small, fusiform, striated and interlacing. They are richly supplied with plexuses of finely divided nerve fibrils ("moniliform fibrils") and with some ganglion cells.[1] The nerves going to the node are found by dis-

[1] Oppenheimer, B. S., and Oppenheimer, A. Nerve fibrils in the sino-auricular node. *J. Exper. M.*, 1912, xvi, 612–619.

section to connect with the terminal divisions of the vagus and sympathetic extracardiac trunks.

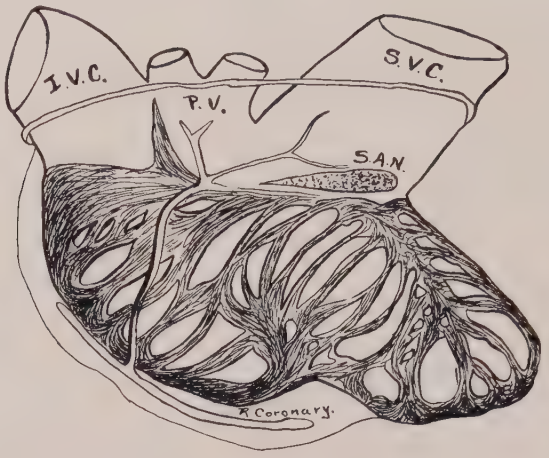

FIG. 3.—Drawing of the right auricle of the dog, to show the chief muscle bands. The sino-auricular node (S.A.N.) is seen situated at the junction of the superior vena cava and right auricle. The precise position of this structure was ascertained by cutting serial sections through tissue taken from the upper part of the sulcus terminalis. (*From Lewis.*)

2. THE AURICULO-VENTRICULAR NODE

This node is situated at the base of the posterior portion of the interauricular septum, near the mouth of the coronary sinus (Fig. 4). Here, the beginning of the node is spread out in a fan-like fashion, interdigitating and thus uniting with the ordinary auricular muscle fibers.

Structurally, it consists of an intricate interlacement of slender spindle-shaped cells which join at all angles. The entire structure is embedded in a network of connective tissue. Nerve fibrils and ganglion cells are scattered in the node and it has a conspicuous arterial supply.

3. THE BUNDLE PROPER

Historically His' bundle was the earliest known tract of communication between the upper and lower chambers of the

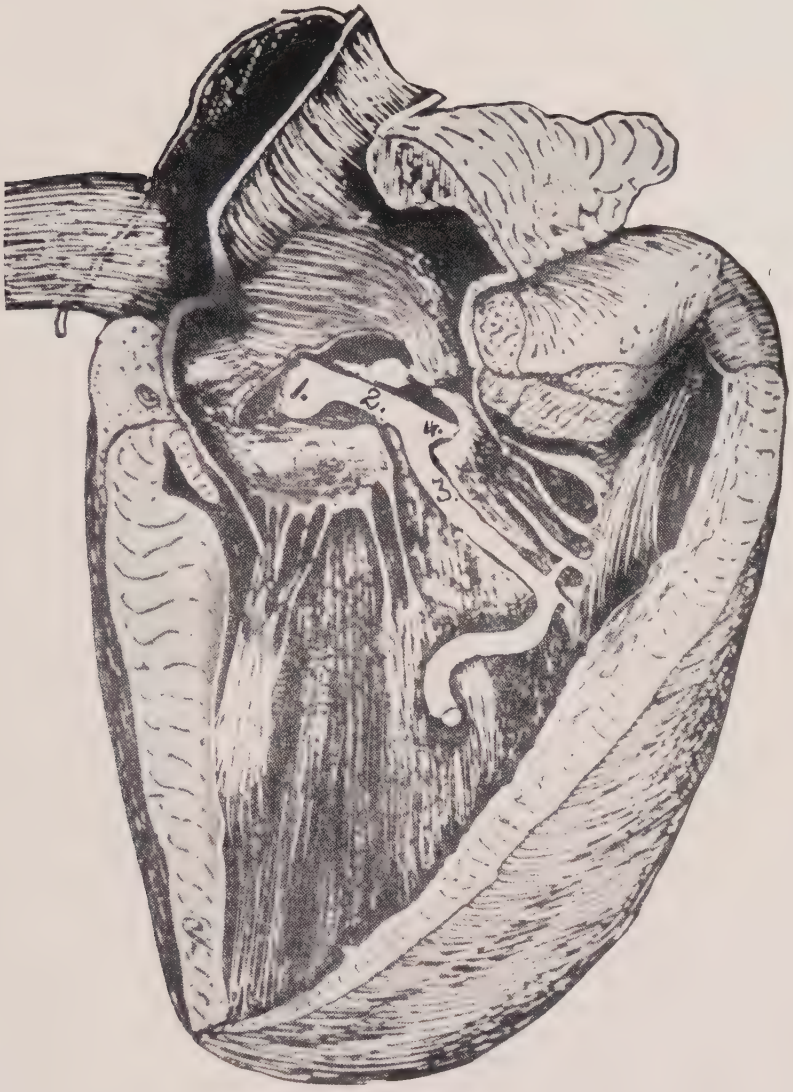

FIG. 4.—Drawing of a preparation in which the A-V junction and the cavity of the right ventricle are exposed to view. It shows the A-V node (1); the bundle of His (2), and the right bundle branch (3). The left branch (4) is shown in part as it pierces the pars membranacea septi. (*After Keith.*)

heart. It begins at the A-V node and is, in fact, a continuation of this node. The bundle runs almost horizontally forward and slightly to the left, pursuing its course to the upper part of the interventricular septum. It is isolated in a fibrous sheath—a special "canal" or "pseudo-bursa." At the anterior part of the "pars membranacea septi" it divides into its two main branches (Fig. 4).

Its fibers are somewhat stouter than those of the A-V node and show a somewhat parallel arrangement.

4. THE RIGHT AND LEFT DIVISIONS OF THE BUNDLE

These divisions begin at the upper part of the ventricular septum. The *left* branch, still sheathed, perforates the membranous septum (Fig. 4) and lies upon the upper border of the true muscular septum; then, curving downward, it enters the subendocardial layer of the left ventricle. It is thin, rather flattened, and fans out promptly into its sub-branches. The *right* branch, also sheathed, continues along the right side of the interventricular septum and soon becomes subendocardial. In contrast to the left branch, this structure is a round, compact bundle and sends few if any branches into the interventricular septum until it begins to divide into its larger sub-branches.

The fibers of these branches are similar to those of the main bundle, except that they are more swollen in appearance and are more sparsely striated.

5. THE TERMINAL ARBORIZATIONS

These arborizations are the finer subdivisions of the right and left bundle branches. The arborizations of the left bundle branch begin upon the septum and are distributed extensively in the subendocardial layers of the left ventricle (Fig. 5).

Some branches terminate in the papillary muscles supporting the mitral valve. The arborizations of the right bundle branch are distributed similarly, some passing through the "moderator band," while others proceed to the papillary muscles supporting the tricuspid valve.

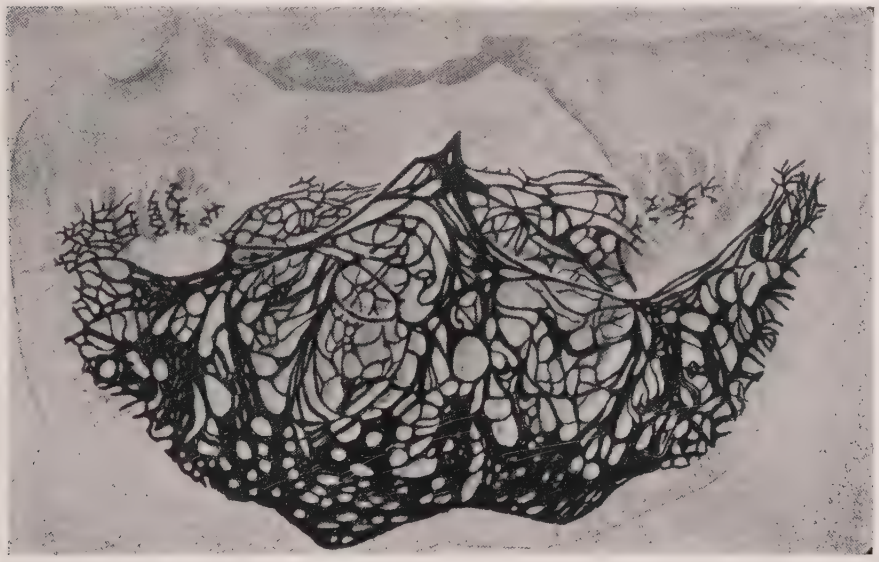

FIG. 5.—Left division of the bundle of His, in the dog's heart, showing the extent of the subendocardial arborizations of the "specific system." (*From Lewis and Rothschild.*)

Some of the ramifications of the right and left main branches, completely sheathed in endocardium, bridge the ventricular cavities and the spaces between the muscular trabeculae. They are referred to as "spurious tendons."

6. THE NETWORK OF PURKINJE

This is a subendocardial network of large, pale and abundantly nucleated specialized cells (Fig. 6). These cells are in intimate relation with the ordinary heart muscle, separated

from it only by a very thin layer of loose connective tissue. They represent the terminal network of the specific system.

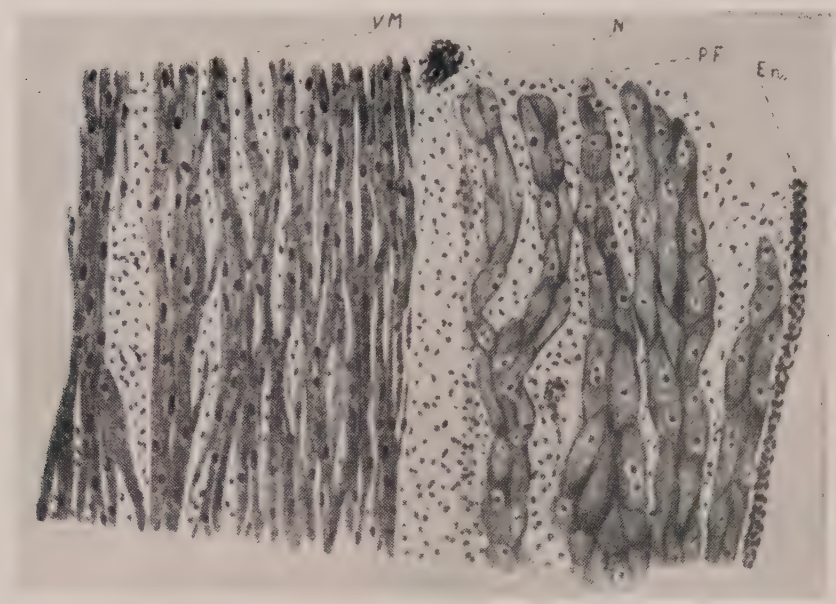

FIG. 6.—A longitudinal section of the moderator band of a sheep's heart (× 70). One of the free borders of the band is seen to the right (En, endocardium). Directly to the left of the endocardium is a number of longitudinally running and branching *Purkinje fibers* (P.F.). These are separated from the ventricular muscle (V.M.) of the band by a loose connective-tissue sheath in which a small portion of a nerve (N) is seen. Note the size of the Purkinje fibers and their large pale nuclei. (*From Lewis.*)

II. PHYSIOLOGICAL PROPERTIES

While the heart muscle proper is concerned chiefly with contraction, the essential function of the specific system is that of stimulus production and stimulus transmission. It is problematical whether the ordinary heart muscle ever initiates a cardiac impulse. On the other hand, there is evidence that the

musculature of the specific system still retains the function of contractility.[1]

Impulse conduction is the property of both, but this function is far more conspicuously developed in the specific system. The relative capacity for transmission of impulse within the heart is approximately in the following order; it is most highly developed in the ventricular division of the specific system (right and left branches of the bundle of His, their arborizations and the subendocardial network of Purkinje). Conductivity in the auricular musculature is next in order, while in the ventricular musculature it is slowest.[2] It is of interest to note in this connection that the glycogen content of these structures varies directly as the rates of their conductivity. In the A-V node transmission of impulse is essentially very slow, as would be expected on account of its structural and chemical peculiarities. The cells of this node are small and intricately interlacing; their glycogen content is relatively low.

THE CARDIAC PACEMAKER

The cardiac stimulus initiating the heart beat is apparently elaborated at the sino-auricular node. For this reason, this node is referred to as the "cardiac pacemaker" or "cardiac pace setter." There are numerous reasons for believing it to be the pacemaker of the heart:

1. MORPHOLOGICAL ANALOGY. The sinus venosus in the lower vertebrate heart is situated between the great veins and

[1] Ishihara, M., and Nomura, S. On the contraction of the branches and terminal ramifications of the auriculoventricular bundle in the heart. *Heart*, 1923, x, 339–403.

[2] Lewis, T., and Rothschild, M. A. The excitatory process in the dog's heart. *Phil. Tr., Lond.*, 1915, s.B., ccvi, 181–226. This article is confined to a study of the excitatory process in the ventricles; but on page 222 there is an excellent summary on the "Excitation wave in the whole heart."

the right auricle. In such hearts, the heart beat is seen to begin in the sinus venosus. The S-A node in the higher mammalian and in the human heart has an analogous anatomical location; in fact, it is held by some that the S-A node is a vestigium of the sinus venosus.[1]

2. THE S-A NODE AS THE SITE OF EARLIEST ELECTRONEGATIVITY. If an electrode is placed over the region of the S-A node and another over any other part of either auricle or great veins, it is found that with the initiation of the electrical changes within the heart, the point of primary electronegativity is always at the S-A node. The earliest evidence of activity is thus found to manifest itself in this region.

3. ELECTROCARDIOGRAPHIC EVIDENCE. Experimental stimulation of any part of the auricles or their appendages yields a sequential electrocardiogram; that is, an electrical curve thus derived shows auricular and ventricular complexes in the proper sequence; but only the one that is forced from the immediate region of the S-A node resembles in all details the *normal* curve for that subject.

4. EVIDENCE OBTAINED FROM WARMING OR COOLING OF THE REGION OF THE S-A NODE. Warming the region of the S-A node raises its metabolic activity and the heart rate is accelerated. Cooling of the region of the S-A node lowers its metabolic activity and a slower heart rate results, showing that conditions which influence the S-A node also influence the heart rate.

[1] "Embryological and developmental studies on the same species and comparative studies of different species indicate that a part of the original sinus tissue enters into the formation of this [S–A node], the remainder becoming condensed into the auricular portion of the A–V node (Koch, Keith, Mackenzie, Külbs, Aschoff)." Wiggers, C. J. Modern Aspects of the Circulation in Health and Disease. Ed. 2, Phila., 1923, p. 26. See also Lewis, T. The Mechanism and Graphic Registration of the Heart Beat. Ed. 3, Lond., 1925, pp. 12–13.

All these facts tend to point to the S-A node as the location where the heart beat is initiated — the structure that sets the pace for the heart. Hence the name, cardiac pacemaker.

SUBSIDIARY CENTERS OF STIMULUS PRODUCTION

The S-A node is the pacemaker of the heart in a relative sense only. It acts as the pacemaker because its rate of stimulus production is faster than that of any other part of the specialized system. It possesses the highest degree of automaticity. If, however, for any reason, the S-A node is impeded, depressed or destroyed, the heart does not, as a rule, stop. It will beat in response to stimuli from some lower level; from some secondary center, usually from the A-V node. If the A-V node is depressed or destroyed, the heart may still beat in response to stimuli generated in the main bundle or in one of its branches.

It is evident, therefore, that, while the S-A node is the cardiac pacemaker, nevertheless the rest of the specific system has the potentiality, under certain circumstances, to set the pace for the heart. In short, any part of this system may be considered a *potential pacemaker.*

Chapter II

Extracardiac Manifestations of the Heart Beat and Methods Employed in Their Study

IN the normal rhythmic heart, similar events recur at regular intervals. Active phases alternate with silent phases. Systole and diastole represent these alternating phases. The dynamics of the intrinsic cardiac mechanism are revealed to us by a group of extracardiac manifestations, some of which are readily detected by our special senses, while others have to be determined by special mechanical aids. It is by means of these peripheral *extracardiac manifestations* that we are made aware of the sequence of events within the heart. It is these that constitute the *clinical signs.*

The extracardiac manifestations of the intrinsic cardiac mechanism are best considered under two general headings, (1) electrical manifestations and (2) hemodynamic manifestations.

1. Electrical Manifestations. They appear in the form of a group of galvanometric deflections in response to a series of intracardiac electrical changes incident to the discharge and transmission of the cardiac impulse and to the heart muscle response. These electrical changes are detected and recorded with the electrocardiograph.

2. Hemodynamic Manifestations. These are the direct result of cardiac contraction and of blood propulsion and are communicated to us as two distinct groups of phenomena: vibratory phenomena, in the form of heart sounds (A); and vascular phenomena, in the form of the arterial pulse (B) and venous pulse (C).

In the following pages, the underlying mechanism, the clinical manifestations and the clinical methods employed in the study of these phenomena are briefly described.

1. Electrical Manifestations of the Heart Beat

The initial manifestation of cardiac activity presents itself as an area of electronegativity at the S-A node, the cardiac pacemaker. From instant to instant this area of electronegativity advances as a wave over the rest of the heart, along a definite, although sinuous and complex path. This *wave of excitation* spreads with great rapidity over the auricular musculature, involving an ever-increasing area. It reaches the A-V node, where it is slightly delayed. From this node, the wave advances, with its greatest rapidity, by way of the bundle of His, the right and left branches of this bundle, their arborizations and the subendocardial network of Purkinje's cells, into the ventricular musculature.

The appearance of this area of electronegativity presupposes a temporarily disturbed electronic equilibrium within the heart, and consequently also the presence of areas of relative electropositivity. As a result, numerous areas with varying degrees of electrical potential differences are set up within the heart.

As the wave of electronegativity (wave of excitation) advances from instant to instant along its definite paths, the magnitude of the multiple potential differences set up by it, as well as the direction and position of their axes, correspondingly vary from moment to moment. The *resultant of the axes* of these numerous areas of potential difference, at any given instant, makes up what is known as the *electrical axis* of the heart.

This resultant, shifting, electrical axis is one of the manifestations of the dynamic events within the heart.

These electrical variations are not confined to the immediate region of the heart, but spread through the body fluids and tissues in all directions, ultimately manifesting themselves as minute electrical potential differences at the periphery of the body. If the terminals of a highly sensitive galvanometer were attached at different points on the surface of the body, in any given plane, these minute peripheral electrical variations which represent, and are in fact a spread of, the intracardiac electrical variations, could be recorded.

The *electrocardiograph* serves just such a purpose. It is a highly sensitive galvanometer which detects and records the minute peripheral electrical variations emanating from the heart during the dynamic phase of the cardiac cycle.

THE ELECTROCARDIOGRAPHIC EQUIPMENT

The electrocardiograph or string galvanometer of Einthoven is based on the well-known physical principle that, *when an electrical conductor carrying current is placed in a uniform magnetic field, it moves in a direction at right angles to both the magnetic field and the direction of the current* (Fig. 7).

The instrument (Figs. 7 and 8) consists essentially of a fiber, of microscopic thickness, made of fused quartz coated with a thin layer of silver or gold, and stretched vertically between the closely approximated poles of a powerful electromagnet. The fiber is protected from dust particles and air currents. Passing through the poles of the magnet and facing each other, separated only by a narrow gap which contains the vertically situated fiber, there are two microscopes, one serving as a condenser and the other as an objective. A beam of light

is passed through these and the magnified and focused shadow of the "string" is projected on a uniformly moving photographic film (sometimes a photographic plate or paper is used.)

When adjusted, with the aid of its accessories, the galvanometer string responds by deflections to the minute

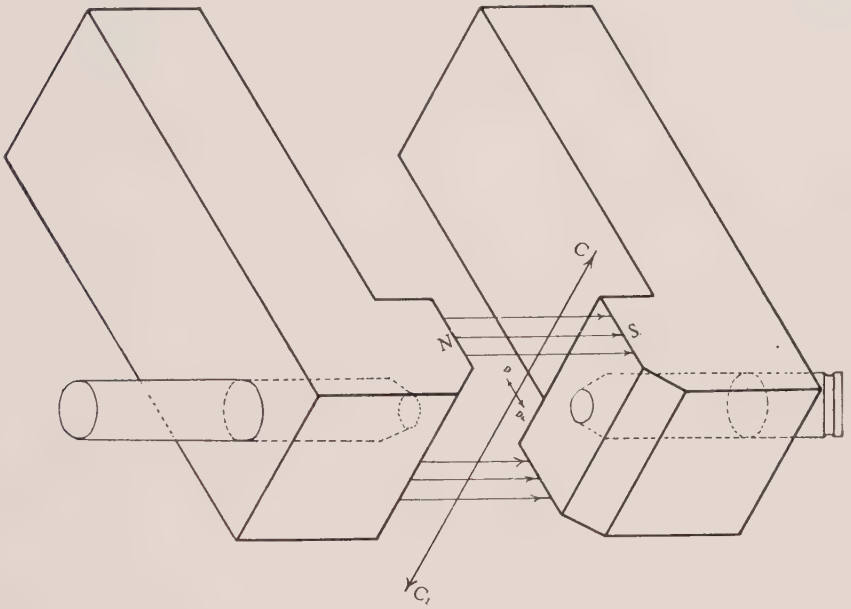

FIG. 7.—Diagram illustrating the principle of the string galvanometer. *When an electrical conductor carrying current* (C-C_1) *is placed in a uniform magnetic field* (N-S), *it moves in a direction* (D-D_1), *at right angles to both the magnetic field and the direction of the current.* In the electrocardiograph the gap (N-S) between the poles of the magnet is essentially very narrow. In this diagram the gap is purposely exaggerated in order that the principles, diagrammatically represented, may be more readily appreciated.

variations of electrical potential at its terminals. The deflections of its magnified shadow are recorded as the "electrocardiographic tracing" or the "electrocardiogram."

The reason for using the Einthoven galvanometer rather than some other form of sensitive electrical instrument is the

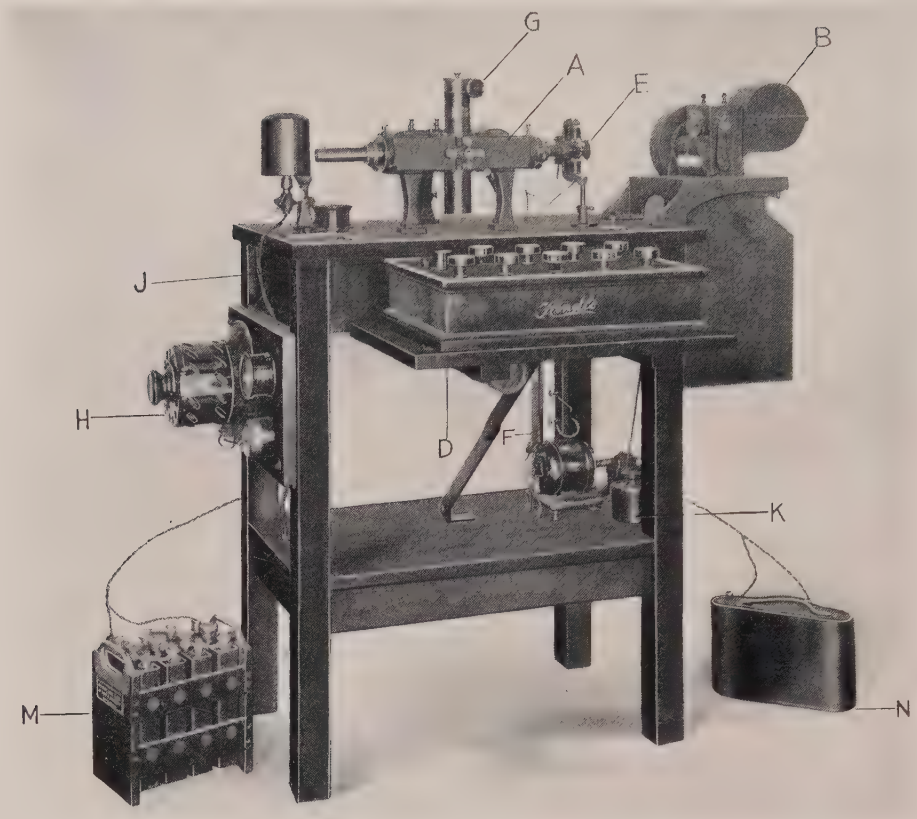

FIG. 8.—Electrocardiograph, American model. A. galvanometer; B. camera; D. resistance box; E. time wheel; F. tuning fork; G. string tension wheel; H. bulb lamp control rheostat; J. galvanometer field switch; K. camera motor control rheostat; M. 6 volt Edison battery; N. 6 volt dry cell.

ability of the Einthoven galvanometer to respond to currents of very short duration.

The following accessories complete the equipment:

1. *A source of light.* A strong, heat-wave-free, straight beam of light from a carbon arc lamp is serviceable. Of late, suitable electric bulbs have been constructed.

2. *A camera, which contains the roll of photo film.* The camera is driven by a small electric motor, the speed of which is adjustable. Within the camera, there is a set of "rollers" carrying the film in a vertical direction, at a uniform speed. The usual speed of the film in clinical cardiography is 25 mm. per second.

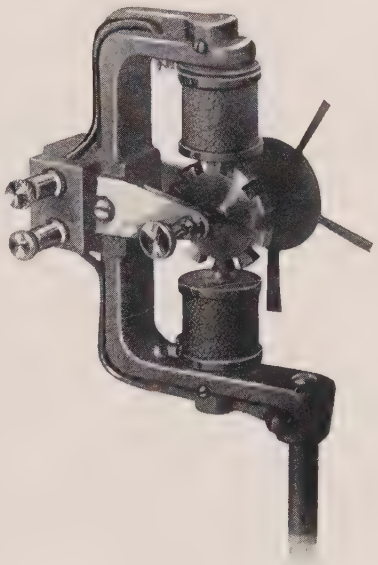

FIG. 9.—Time marker.

3. *A time marker.* Any device that interrupts the light at the desired regular interval suffices. The one most useful is the "episkotister" (Fig. 9). This is a device in which a wheel with five spokes (one heavy and four finer ones) revolves in the path of the beam of light going to the camera. The uniformity of rotation of this wheel is maintained by an electrically driven tuning fork or reed. As each complete revolution occupies one-fifth of a second and since there are five spokes to the revolving wheel, the beam of light is intercepted twenty-five times each second. These interruptions are recorded on the running film, as lines corresponding to the number of spokes that intercept the light per second. The heavy spoke, therefore, records one-fifth second and the fine ones, one-twenty-fifth second.

4. *The compensating "resistance box."* This accessory holds a dry cell battery, an ammeter, a switch, pole changer and a group of resistances which are shunts to the main circuit. It is by the aid of these that we are able to use the main circuit under proper control, that is, "compensate" for the undesirable body current and "standardize" the tension of the galvanometer string to a desired degree of sensitivity.

TECHNIQUE OF ELECTROCARDIOGRAPHY

The subject to be electrocardiographed is placed in circuit with the galvanometer by means of suitable electrodes applied

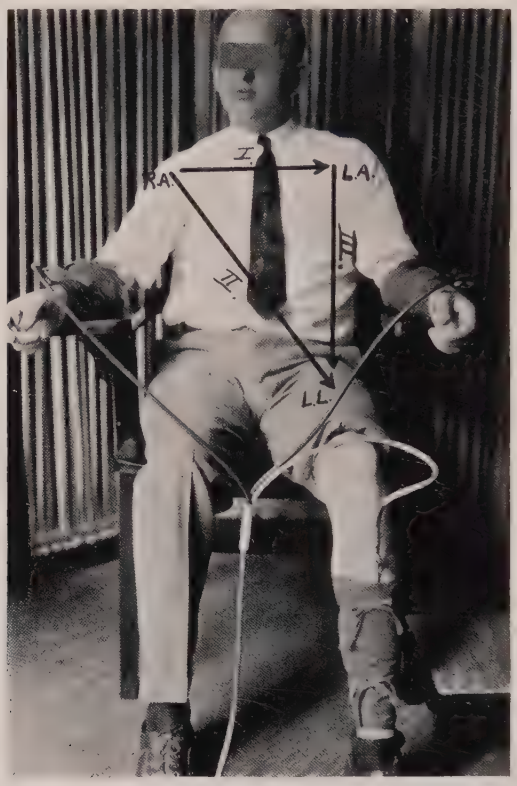

FIG. 10.—The subject is connected to the electrocardiograph and thus introduced into the circuit with the recording galvanometer, by three conventional "leads." In the photograph, three wires, carried by a common cable, are seen attached to electrodes applied to the right arm, left arm and left leg, respectively. The "lead" resulting from the combination of any two of these wires is indicated at the sides of a triangle drawn upon the figure. (R.A.→L.A = lead I; R.A.→L.L. = lead II; L.A.→L.L. = lead III.)

to the body surface, so that the minute peripheral electrical variations incident to the heart action may be conducted to the

instrument with the least possible distortion (in routine clinical work, the electrodes are applied to the extremities Fig. 10). Three different "leads" are employed and a record is taken with each of these leads in turn. The customary leads in clinical work are:

Right Arm ⟶ Left Arm = Lead I
Right Arm ⟶ Left Leg = Lead II
Left Arm ⟶ Left Leg = Lead III

With the subject in circuit, lead chosen, magnet excited, light adjusted and time marker running, we proceed to take a record, the electrocardiogram.

However, at this stage the galvanometer is not yet influenced by the subject, because it is guarded by a protecting rheostat in the main circuit. It is evident that the use of a "protecting" rheostat implies caution. But, caution against what?

When a subject is in circuit with the galvanometer and the circuit closed, the instrument is influenced in two ways.

The Constant Current or Skin Current. The first influence is in the form of a *constant current* which deflects the string and tends to keep it deflected as long as the subject is in circuit. The deflection is due to a constant potential difference offered by the two points of the body surface at which the electrodes are applied. The activity of the sweat glands over the body surface is presumed to be the origin of this potential difference, and the current thus generated is often called the *skin current.*

Such constant current is at times strong enough to cause so marked a deflection as to throw the string shadow to one side and out of range of the recording camera; at times quite out of the operator's view. The deflection may be so violent as to break the delicate galvanometer string. Hence, the caution

(already mentioned) of introducing a protecting resistance, which enables the operator to bring the subject into the circuit gradually, and permits him to neutralize this undesired current, step by step, in a manner to be described below.

The Fluctuating Current. The second factor influencing the galvanometer appears in the form of a *fluctuating current*, resulting from intracardiac electrical variations incident to the dynamic phase of the cardiac cycle. In contradistinction to the skin current, which, as we have seen deflects the string to one side and keeps it deflected, the intracardiac electrical variations manifest themselves as periodic groups of deflections of the galvanometer string. These constitute the *electrical manifestations of the heart beat.* It is these deflections that we wish to record; they yield the electrocardiographic tracing, the electrocardiogram.

"Compensation" and "Standardization." Before we can record these deflections, we must pay attention to two points: First, we must remove the undesired direct body current. Secondly, we must adjust the sensitiveness of the galvanometer string to a standard, so that it will deflect in response to a given potential difference at its terminals with a standard amplitude of deflection.

Compensation. Removal of the direct current is accomplished by *compensation*, or *neutralization*, in the following way: The subject is introduced into the circuit gradually, by means of the protecting rheostat, thus permitting only a fraction of his skin current to enter at each step.[1] As the string deflects in response to these minute direct currents, an opposing electro-

[1] As a rule the protecting rheostat consists of three degrees of resistance: "infinity" (circuit broken), 100,000 ohms' resistance and 10,000 ohms' resistance. When the instrument is not in use, the rheostat is kept at infinity. When patient is first introduced into

motive force, of suitable magnitude, derived from a shunt, is allowed to enter the circuit to compensate for it or neutralize it.

In the shunt or compensating circuit (Fig. 11) there is a dry cell battery, switch, pole changer, variable resistance (R_2) and a microammeter. The compensating current issues from the battery, goes through the switch and its direction is determined by the pole changer. The variable resistance

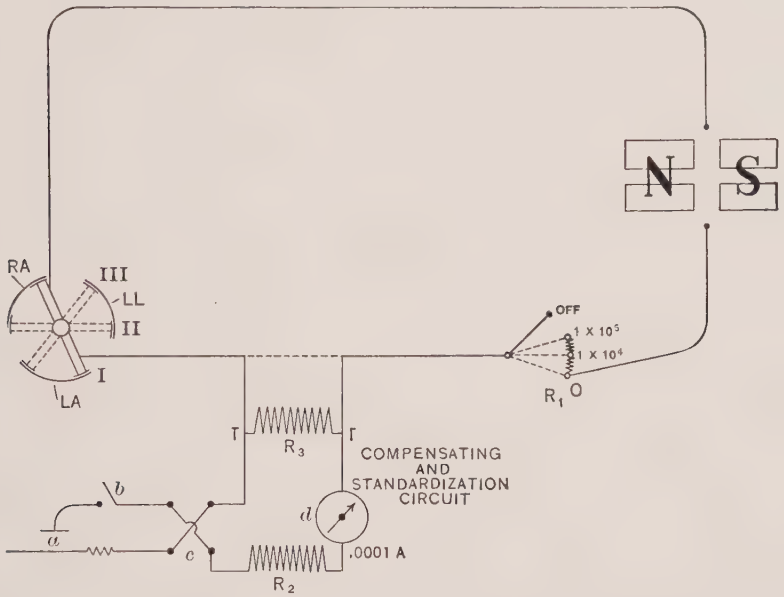

FIG. 11.—Scheme showing connection of patient with galvanometer and also Einthoven's method of compensation and standardization, as adapted to the American model of the string galvanometer. RA, LA, LL, selecting switch to attach patient; R_1 = protective rheostat; R_2, = rheostat controlling output of compensating current; R_3, = rheostat for standardization and compensation. *a*, battery; *b*, switch; *c*, pole changer; *d*, microammeter. (*From Wiggers.*)

(R_2) is adjusted to a constant so as to establish a standard current of 0.0001 amperes. This is indicated by the microammeter. Another variable resist-

the circuit, this infinite resistance protects the galvanometer string from a sudden introduction of a large potential difference at its terminals. The circuit is opened by interposing the above mentioned resistances in successive steps and the patient's body potential difference is compensated at each step. Finally when all resistance has been removed and patient's skin current compensated, there remains only the fluctuating cardiac current to influence the galvanometer string.

ance (R_3), also a part of the compensating circuit, is so adjusted that a variation of 10 ohms resistance in its path permits a potential difference variation of one millivolt in the main circuit containing the patient and the galvanometer. It is this derived one millivolt per step, using as many steps as necessary, that is utilized in compensating or neutralizing the constant potential difference offered by the two points over the body surface at which the electrodes are attached.

Standardization is necessitated by the fact that if we wish to compare different curves, that is, if we wish to evaluate the amplitudes of corresponding deflections in different curves, we must assure ourselves that the instrument we use is standard; namely, that the galvanometer string is adjusted to a standard degree of sensitivity. The standard originally adopted by Einthoven is now generally used. It requires that the tension of the string be so adjusted that *its shadow, measured on the film, deflects one centimeter for each millivolt* of potential difference applied to the circuit containing the galvanometer and the patient. The compensating circuit which, as we have seen, is adjusted to a potential difference variation of one millivolt per step, serves also as the standardizing circuit. In standardizing, therefore, we introduce one millivolt of potential difference during diastole and note the deflection of the string shadow at the camera slit. If the deflection is less than one centimeter, the string is loosened; if more, it is tightened. The operation is repeated until the desired standard tension is obtained.

Compensation and standardization must be repeated for each of the three leads separately.

Determination of Patient's Resistance by "Comparison." After the electrocardiogram has been taken, it is sometimes desirable to ascertain how much resistance the patient offered to the main circuit, because high resistance appreciably alters the quickness of response of the galvanometer string. If

very high, it may actually distort the electrocardiographic tracing and thus render it unsuitable for detailed study.

The patient's resistance is determined by "comparison" in the following way: The control box (resistance box) holds a set of variable resistance coils, calibrated in units of 1000 ohms and 100 ohms. After the patient has been removed from the galvanometer circuit, introduction of a millivolt of potential in order to test the sensitiveness of the string shows that its deflection now differs appreciably from the standard to which it was adjusted when the patient was still in circuit. The string shadow at the camera slit is now seen to deflect with a greater amplitude than one centimeter per millivolt; it may now deflect two, three or more centimeters, indicating that the patient, while in the circuit, has been a restraining influence, a source of resistance. The degree of such resistance is ascertained by substituting, in place of the patient, several units of the resistance coils above mentioned. This is done in successive steps, testing the string sensitiveness at each step. When the deflection of the string again corresponds to the standard amplitude, it is assumed that the *patient's resistance* has been fully substituted and that, *by comparison*, it is *equal to the number of ohms* that was required to bring the galvanometer string back to its original standard deflection.

THE NORMAL ELECTROCARDIOGRAM

The electrocardiographic tracing (Fig. 12) is a graphic expression of the peripheral manifestations of a series of intracardiac electrical changes, derived in a single plane—the coronal plane of the body.[5] It consists of a group of characteristic

[5] While intracardiac electrical variations spread all over the body, and, as has been stated, can be detected in any plane, the clinical electrocardiogram as ordinarily derived does not represent all of its manifestations. It merely represents a "manifest value" derived in the frontal or coronal plane.

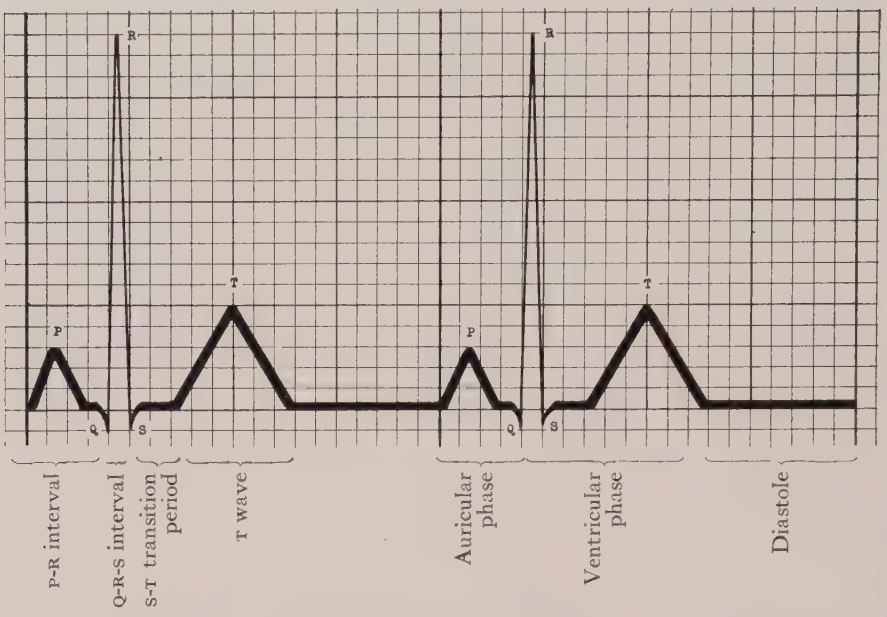

FIG. 12.—Diagram of a normal electrocardiogram drawn to scale. Two cardiac cycles are represented. The complexes are designated by the letters P,Q,R,S,T. The various *intervals* are indicated beneath the first cycle (left). The auricular and ventricular phases and the period of diastole are indicated beneath the second cycle (right). Vertical lines in background of diagram represent time: heavy lines = one-fifth second; fine lines = one twenty-fifth second. Horizontal lines designate height of waves in millimeters.

waves which different investigators have designated by various names. The terminology of Einthoven, the alphabetical designation P, Q, R, S, T, has, however, been almost exclusively adopted.

These waves are often referred to as being either upright or inverted. The terms simply mean that these waves represent deflections of the shadow of the galvanometer string either to the right or to the left: and since the tracings are generally examined as horizontal strips, the right deflections appear upright and the left ones appear inverted. Some use the terms positive and negative instead of upright and inverted.

Characteristic Configurations and Time Relations of the Various Components of the Electrocardiogram.

The P wave, is a blunt or slightly peaked upright wave.

The Q wave is a small, sharp, inverted wave (inconstant).

The R wave is a tall, slender, sharply pointed and clearly outlined upright wave.

The S wave is a small, sharp, inverted wave (inconstant).

The T wave is generally a blunt, widely curved, upright wave.

The P-R interval is the period from the beginning of the P wave to the beginning of the R wave. It measures normally from 0.12 to 0.20 seconds.

The Q-R-S interval, measured between the foot-points of the Q, R, S complex, varies between 0.04 and 0.08 seconds. A duration of 0.10 seconds is regarded as the upper limit of normal.

The S-T interval is a horizontal (isoelectric) or nearly horizontal line of variable length, extending from the S wave to the beginning of the T wave. The designation, S-T transition period is sometimes employed.

Significance of the Components of the Electrocardiogram:

The P wave: The beginning of the P wave corresponds to the beginning of the auricular activity.

The P-R interval expresses the time elapsing between the beginning of auricular and the beginning of ventricular activities. It marks the auriculoventricular conduction time and includes the normal physiological delay at the A-V node.

The Q, R, S, T group of waves constitutes the ventricular phase of the electrocardiographic record. These waves are related to ventricular systole.

The Q, R, S complex is referred to as the initial ventricular deflection. It is regarded as corresponding to the time of spreading of the stimulus through the ventricles.

The S-T interval corresponds to the period of "holding of the stimulus"—active ventricular contraction.

The T wave, also designated as the terminal ventricular deflection, corresponds to the "receding of the stimulus," completion of the ventricular phase. This view is not accepted by all.

The Electrocardiogram as a Source of Information on Cardiodynamics. The electrocardiogram is a graphic representation of a series of electrical manifestations occurring in the course of the dynamic phase of the cardiac cycle. When auricular and ventricular complexes show normal configurations, and the intervals designating conduction time are within normal limits, the curve serves as a record of the *normal* sequence of intracardiac events. It implies that the cardiac impulse is elaborated, built up and discharged at the normal site of the pacemaker, and that the cardiac impulse is conducted unhampered along its normal pathways.

Impairment at the site of impulse production, or in the course of the pathways of the stimulus, is shown in the graph, either as an aberration in the form of the waves, or as an alteration in the lengths of the various isoelectric intervals. Clinically such disturbances usually appear as arrhythmias and the electrocardiographic records enable us to interpret the underlying disturbance of the intrinsic cardiac mechanism giving rise to the clinical irregularity. In fact, it is in this group of disorders that the electrocardiogram contributes its greatest usefulness.

II. Hemodynamic Manifestations of the Heart Beat

A. HEART SOUNDS

While the human ear is far from being a perfect instrument for the detection and differentiation of fine variations in sound vibrations, it can nevertheless often be improved by diligent training. Some clinicians, especially those of the older schools, who have had to rely largely upon their special senses to aid them in their diagnostic efforts, have often developed them to considerable degrees of acuity. In recent years, especially since the advent of instruments of precision, we are inclined to rely less upon our special senses and incidentally fail to make the necessary effort to develop them. Since, however, circumstances under which the clinician is still compelled to work are far from the ideal and since instruments are not always available and, particularly, since it is the man and not the instrument who ought to be the final judge, the potentialities of our special senses as simple, useful and readily available means in clinical diagnosis ought to be developed and utilized.

In the case of heart sounds, if a physiological investigation is the object in view, instruments are desirable and, in the fact, necessary; but, at the bedside, we are still forced to rely upon

our auditory apparatus, aided perhaps by the stethoscope. In the study of arrhythmias, auscultation is generally helpful because in these disorders we are concerned with but simple changes in the characteristics of heart sounds such as, alterations in pitch, intensity, duration and rhythm.

Since an intelligent interpretation of any clinical disorder of function presupposes at least a fundamental knowledge of the normal, therefore, in order to appreciate the aberrant forms of heart sounds, an understanding of the dynamic events that transpire during a normal cardiac cycle, emphasizing especially those factors which make up the components of the heart sounds, becomes an essential prerequisite.

The following is a brief description of the mechanism, clinical characteristics and some of the methods employed in the study of the normal heart sounds:

Mechanism. During the latter part of the diastolic phase of the cardiac cycle, the phase known as the "period of diastasis," blood flows passively from the great veins into the auricles and from there into the relaxed ventricular cavities. Meanwhile the cardiac impulse is being built up and is rapidly approaching its critical point. Having reached it, it is discharged, and the dynamic phase of the cardiac cycle is initiated. The auricles contract and forcibly eject their contents into the ventricles. Auricular relaxation follows. By this time, the impulse has spread to the A-V junction, and after a slight delay in this region, it spreads onward again, this time with great rapidity, through the ventricular division of the specific system.

At this stage of the cardiac cycle, ventricular filling is maximal and the auriculo-ventricular valves are in apposition.

When the excitation wave, having advanced by way of the ventricular division of the specific system, reaches the ven-

tricular musculature, ventricular systole begins. The ventricles contract upon their contents—an incompressible fluid— causing a sudden, sharp rise of pressure within their cavities, and a marked tension and *vibration of the musculature of the ventricular walls.*[1] As a result of the increasing intraventricular pressure incident to systole, the auriculo-ventricular valves are forced in the direction of the auricles, but they are at the same time held taut by the chordae tendinae, so that *the auriculo-ventricular valves vibrate* between these two forces. The rising pressure within the ventricular cavities soon exceeds the pressure within the aortae, and, therefore, the semilunar valves are forced open and the *ventricular contents are forcibly ejected into the arterial tree.*

Subsequent relaxation of the ventricles follows. This permits a rapid fall of intraventricular pressure which for a moment becomes equal to, but soon falls below the pressure within the roots of the aortae just injected. The direction of the pressure is thus reversed, leading to a *sudden, forcible closure and vibration of the semilunar valves.* The systolic phase of the cardiac cycle is completed. Diastole again supervenes.

Physiological Components and Clinical Characteristics of the First and Second Heart Sounds. Contraction and vibration of the ventricular musculature, vibration of the taut auriculo-ventricular valves and the rushing of the blood column into the aortae make up the components of the

[1] Contraction of the ventricular musculature is sudden and forceful: it is accomplished *almost* simultaneously throughout the entire substance of both chambers. The arrangement of the long strands of the ordinary heart muscle groups in intertwining layers cannot account for, and in fact does not favor, such a mechanism. It is the peculiar architecture of the ventricular division of the specific system, namely, the extensive distribution of its terminal arborizations, stimulating a great number of different areas of the ventricular myocardium at the same time, that enables the sudden forceful and almost simultaneous contraction of both ventricles.

first heart sound. The sudden and forceful closure of the semilunar valves and their brief vibration make up the components of the *second heart sound.*

The first heart sound is longer and of a lower pitch than the second. It is preceded by a long pause and followed by a short pause. It coincides with the height of ventricular systole and is predominantly muscular in character.

The second heart sound is shorter and is of a higher pitch than the first. It is preceded by a short pause and is followed by a long pause. It marks the beginning of diastole and is predominantly valvular in character.

The Third Heart Sound. A normal third heart sound is described by many as occurring shortly after the second—a sort of echo of the second heart sound. It occurs not sooner than 0.12 second following the second heart sound. It does not seem to be a simple reduplication of it. The cause of the third heart sound is not quite clear and is, therefore, ascribed to various causes by different observers. Some attribute it to vibrations of the auriculo-ventricular valves in early diastole, when, upon relaxation of the ventricles and opening of the auriculo-ventricular valves, the accumulated blood in the auricles suddenly falls into the relaxed ventricular cavities, causing those valves to float and vibrate in the current. Suggestions have been made that it may be due to auricular systole of the subsequent cycle. The time of its occurrence and the nature of its recorded vibrations do not favor the probability of such origin.[1] Other causes are suggested, most of which, however, seem untenable. The clinical significance of the normal third sound is negligible. It is but rarely heard clinically except when very marked and becomes the component of a gallop rhythm. The sound is most often found in heart sound records—phonocardiograms.

Clinical Methods Employed in the Study of the Heart Sounds. The simple means of direct auscultation or auscultation with the aid of the stethoscope generally yields considerable information regarding the clinical characteristics of the first and second heart sounds and their time relation to

[1] Third heart sounds, due to auricular systole (with prolonged P-R interval), have been demonstrated by H. Mond and E. M. Oppenheimer (personal communication).

the rest of the cardiac cycle. At any rate, the information thus gained is generally sufficient to enable one to recognize ordinary alterations in the characters of the heart sounds as well as ordinary changes in time relations that the heart sounds bear to one another and to the rest of the extracardiac manifestations of the heart beat.

For a more detailed study of the vibration frequencies and the accurate time relations of the heart sounds, as well as for the study of cardiac murmurs, graphic methods are employed. Of these, but two will be described.

1. *The Electrical Method* (*The Einthoven Microphone*). This apparatus is essentially a carbon cup microphone not unlike that used in the telephone receiver (Fig. 13). It is enclosed in a sound-proof box so as to exclude extraneous noises. The heart sounds are led to it from the chest wall by means of a semi-rigid rubber tube, at the far end of which a cup or funnel is attached to serve as a chest piece.

The microphone receiver is in circuit with a dry cell battery, a suitable rheostat and a primary coil. Near this primary coil there is a movable secondary coil which in turn is in circuit with a string galvanometer. The sound vibrations from the chest are transmitted to the diaphragm of the carbon cup, causing it to oscillate. Oscillations in the microphone cause corresponding electrical variations in the

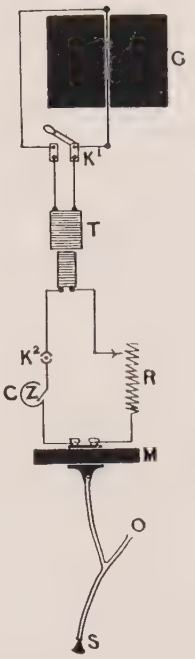

FIG. 13.—A diagram illustrating the apparatus (Einthoven Microphone) used in taking heart sound records. M. Microphone. S. Stethoscope end-piece. O. Tube outlet. K^2. Make-break key. R. Rheostat. T. Transformer. K^1. Short-circuiting key. G. String galvanometer. (*From Lewis.*)

primary coil which in turn induces electrical variations within the secondary coil near it. The galvanometer string, in circuit with the secondary coil, deflects in response to these minute electrical variations. The deflections are recorded on a moving film as the heart sound record (Fig. 14). The

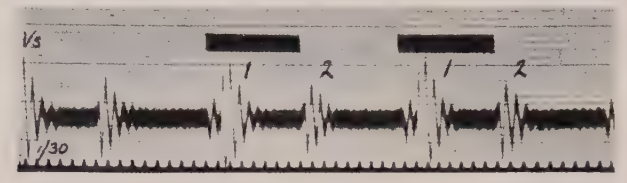

FIG. 14.—Record of normal apical heart sounds, taken by the electrical method. (*From Lewis.*)

apparatus is somewhat bulky, costly and requires a specially trained technician.

2. *The Optical or Direct Method* (*The Frank Capsules*). This direct method endeavors to reproduce sound vibrations not unlike that reproduced by the human ear. The sound vibrations are collected by means of a well-fitting cup or funnel chest piece and transmitted by means of a semi-rigid rubber tube to a fixed "capsule." This is a small modified tambour over the end of which there is a tightly drawn, very sensitive rubber membrane or rubber film. The fine membrane or film vibrates in response to air vibrations in the transmitting tube, thus simulating the human membrana tympani. A tiny mirror is fixed to the membrane in an excentric position. The vibrations imparted to the membrane cause the mirror to oscillate at a corresponding rate. A narrow beam of strong light is focused on the oscillating mirror which thus reflects an oscillating beam of light onto a moving photo film (Fig. 15).

B. THE ARTERIAL PULSE

The arterial pulse has long been regarded by clinicians as a readily available index to cardiac dynamics. In fact, certain types of pulse beats have become known as characteristic expressions of certain hemodynamic disturbances. They are

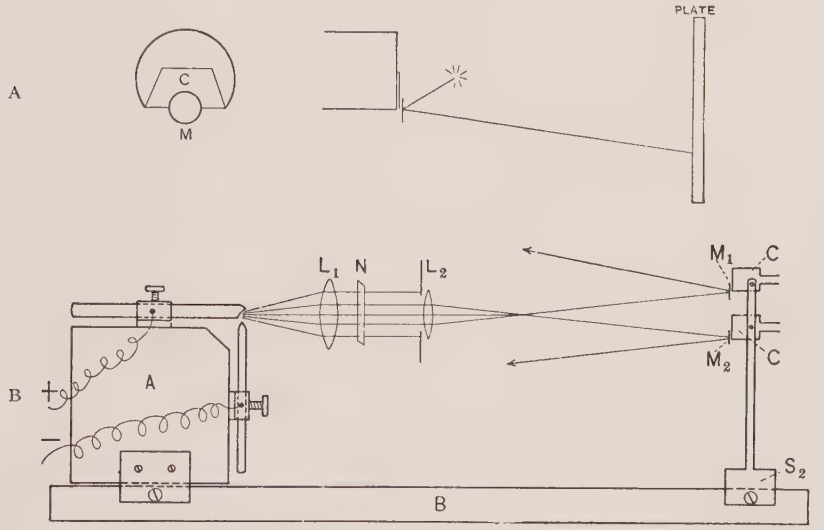

FIG. 15—A. Diagram showing principle of segment capsule (C), mounting of mirror (M) and the method of reflecting light beams to sensitive plates. B. Diagram of optical illuminating system for reflecting light from segment capsules. A, arc light, movable on a solid bench B; rays from positive carbon pass through a condensing lens (L_1) and an adjustable slot (N), the image of which is focussed by a lens (L_2) and projected upon the mirrors (M_1 and M_2) of the segment capsules (C). These capsules are supported on a vertical rod sliding by a clamp (S_2) upon bench (B). Of the two mirrors illustrated (M_1 and M_2), while one is used for taking a heart sound record, the other may be used for a synchronous pulse tracing. (*From Wiggers.*)

thought to be especially indicative of the presence of certain valvular defects; hence, the terms, "aortic pulse" in aortic insufficiency, the "pulsus tardus, rarus et parvus" in aortic stenosis, etc.

In the cardiac irregularities, estimation of the pulse beat has not been utilized as a clinical index to its fullest possibilities. In the past, the term "irregular pulse" often covered a multitude of uncertainties regarding the actual significance of clinical variations in pulse volume and rhythm. The epoch-making work of Mackenzie[1] brought new light to those who were seeking further information, especially to those who were ready and willing to pursue graphic studies. Such studies rendered the meaning of pulse changes comprehensible. The average clinician, it seems, has not gained proportionately, however, for he does not as a rule avail himself at the bedside of all the accumulated information. This is not at all surprising however, because the appreciation of the significance of abnormal pulse variations necessitates an understanding of the altered cardiac mechanism responsible for their production and the importance of such knowledge has not been sufficiently impressed upon the clinician.

On the other hand, it is equally evident that an understanding of the abnormal requires at least a cursory knowledge of the fundamental factors that comprise the normal.

With this point in view, the following chapter is offered as a brief description of the mechanism, mode of transmission and clinical characteristics of the normal arterial pulse. For the sake of completeness, the principles of some of the graphic methods employed are included.

Mechanism and Clinical Characteristics. When the ventricles contract during systole, and expel their contents into the arteries, an added volume of blood is accommodated in the arterial tree. If the arteries were rigid tubes, this added volume

[1] Mackenzie, J. The Study of the Pulse, Arterial, Venous, and Hepatic, and of the Movements of the Heart. Lond., 1902.

of inelastic fluid, transmitting pressure in all directions, could be accommodated only by a complete forward movement of the entire blood column. Since, however, the arteries are normally highly distensible and elastic, such accommodation is accomplished largely by a distention and elongation of the arterial walls, for it evidently requires less pressure to distend them than to move the entire blood column forward. The arterial distention naturally manifests itself, first and most markedly, in the vessels nearest to the heart, namely, in the aortic roots, whence it progresses peripherad with great rapidity, ultimately spending itself in the finer arterioles and arterial capillaries. *This distention first manifested at the root of the aorta, as a result of the increased pressure imparted to it by each successive ventricular systole, passes along the arterial tree, from point to point, as the Arterial Pulse Wave.*

The velocity of this pulse wave is independent of the velocity of the blood flow. The pulse wave is in advance of the blood flow and, in fact, aids in the accommodation of the advancing blood column which, in part at least, is propelled ahead by the successive elastic recoil of the previously distended arterial segments. The pulse wave advances 6 meters to 10 meters per second, while the blood flow rarely exceeds a velocity of one-third meter per second.[1]

The arterial pulse presents certain *clinical characteristics* which are in accordance with its mode of genesis and transmission. On palpation, and still more prominently in graphic records (Fig. 16) it is noted to *rise suddenly*, due to the sudden force imparted to it by ventricular systole, and to *decline more gradually*, as a result of the elastic recoil of the arterial walls.

[1] Wiggers, C. J. Modern Aspects of the Circulation in Health and Disease. Ed. 2, Phila., 1923, p. 190.

In graphic records it is seen that, before its complete decline and often near the middle of its descending limb, a small secondary wave, the dicrotic wave, appears.[1]

The *rate* and *rhythm* of the normal pulse coincide exactly with the rate and rhythm of the heart beat. The *size* of the pulse depends largely upon the force of ventricular contractions and upon the volume of blood ejected with each systole; in part, it also depends upon the elasticity and the degree of

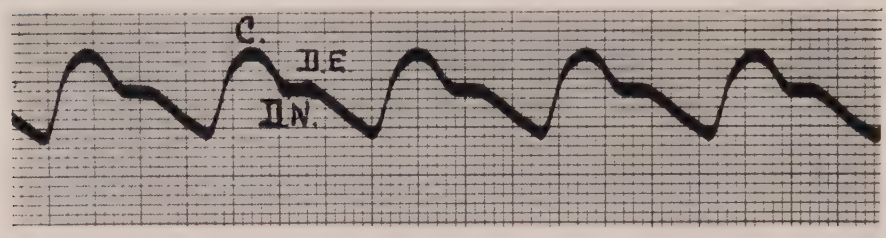

FIG. 16.—Tracing of an arterial pulse (brachial) to show the sudden rise (C, crest) and gradual decline of the arterial pulse wave. The dicrotic notch (D.N.) and dicrotic elevation (D.E.) are seen on its descending limb.

vasomotor stability of the arteries. Accordingly, if a heart beat is premature as a result of which the systolic output may be diminished, the corresponding pulse beat may be smaller than that which is normal for the same subject. At times, such beat may be too feeble to force open the semilunar valves and therefore may induce no arterial pulse. On the other hand, a cardiac pause (beyond the normal diastolic pause for a given subject)

[1] Besides the main crest, an arterial pulse tracing generally shows also a small notch followed by a small crest. These are situated on the descending limb of the main pulse wave and are designated as the dicrotic notch and the dicrotic wave respectively. Simultaneous tracings of intracardiac and aortic pressure changes show that the dicrotic notch is synchronous with the closing of the semilunar valves. It is assumed, therefore, that the sudden temporary yielding of these valves at the moment of closure is responsible for the slight negative wave, the dicrotic notch; and that their prompt recoil, immediately following, is the cause of the dicrotic wave.

because it permits a greater cardiac filling and consequently greater systolic output, may give rise to an unusually large pulse beat in the cycle immediately following it.

The description of the various types of arterial pulse changes as a result of valvular defects is not within the scope of this book. We are rather concerned with alterations in rate, rhythm and size of the pulse. It is our purpose to stresss those changes which may aid us in the clinical recognition of the cardiac irregularities.

Methods Employed in the Study of the Arterial Pulse. Various mechanical devices are in use to record and study the arterial pulse; but since, as has been stated, this text is concerned largely with the rate, rhythm and size of the pulse and not with the minute details of its configuration, such devices will be but briefly described. Those desiring more information are referred to standard texts on the subject.[1]

The following general types of mechanical devices are used.

1. The type in which the arterial pulsations are transmitted by means of a button, spring and writing lever to a uniformly moving paper (Dudgeon Sphygmograph).

2. The type in which the arterial pulsations are transmitted by means of a button and spring to a tambour, whence, by means of a rubber tube the pressure variations reach a second smaller tambour. Writing levers attached to the small tambour record the pulsations on a uniformly moving paper. (Mackenzie Ink Polygraph.) (Fig. 17.)

[1] Mackenzie, J. The Study of the Pulse, Arterial, Venous, and Hepatic, and of the Movements of the Heart. Lond., 1902.

Lewis, T. Mechanism and Graphic Registration of Heart Beat. Ed. 3, Lond., 1925.

Wiggers, C. J. Modern Aspects of the Circulation in Health and Disease. Ed. 2, Phila., 1923.

3. The type in which the arterial pulsations are transmitted by air in a semi-rigid rubber tube to a "segment capsule" and recorded by the direct optical method. (See direct optical method described for heart sounds pp. 36 and 37.)

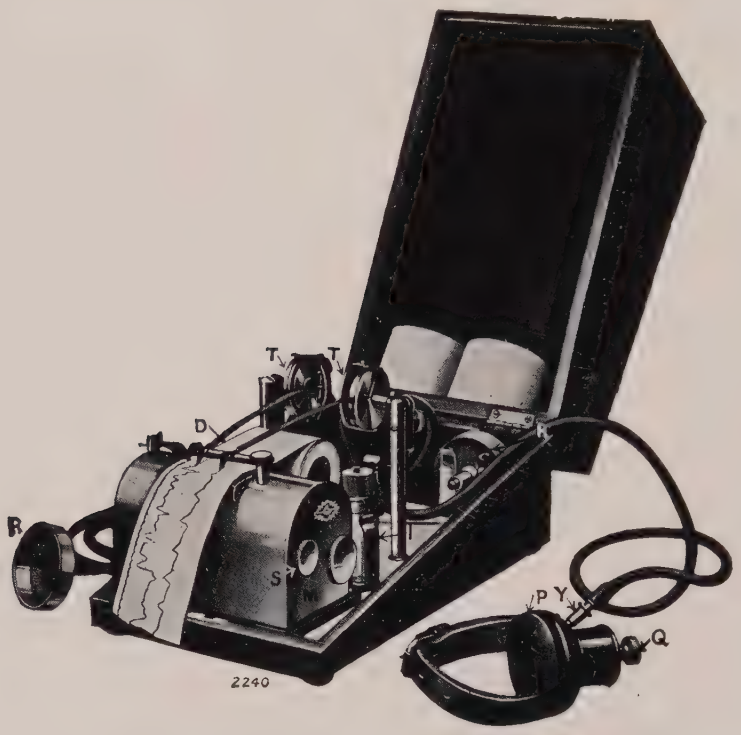

FIG. 17.—The Mackenzie-Lewis polygraph. I, ink bottle; M, aluminum case containing clock-work movement; P, glycerine pelotte; Q, adjustment for altering tension on strap; R, venous receivers; S, arrangement for varying paper speed; T, tambours; Y, valve.

For purely clinical purposes, sufficient information can be gained by the ordinary method of palpating the superficial arteries. Palpation yields readily available information regarding the rate, rhythm and transitory variations in size of the arterial pulse.

C. THE VENOUS PULSE

The clinical features of the venous pulse have been thus far but rarely utilized as aids in diagnosis, except by those who have acquired the technique of polygraphy.

As a matter of fact, the mere visualization of the venous pulse, the jugulars, may at times yield valuable information not available by any other means. Because of their superficial situation and their visible pulsation, one may, with little training, learn to use them as helpful adjuncts in clinical diagnosis of the cardiac irregularities.

The following is a brief description of the mechanism, clinical characteristics and methods employed in the study of the normal venous pulse.

Mechanism and Clinical Characteristics. Since the arterial pulse wave disappears in the terminal divisions of the arterial tree, it cannot, under normal circumstances, be transmitted into the venous system. It cannot, therefore, contribute to the formation of the venous pulse. The venous pulse has an entirely different mechanism.

On visualization of the external jugular vein or jugular bulb (Fig. 18), two phenomena may be observed. On the one hand, the veins are seen to collapse during inspiration and to distend during expiration. This is due to alternating intrathoracic pressure changes incident to respiration. On the other hand (especially visible at the end of expiration), the jugulars show a fine oscillating wave which consists of multiple components. This is the true venous pulse and is caused by intracardiac pressure changes.

Ordinarily, the venous pulse can be visualized in many instances if the subject is placed in proper position and in

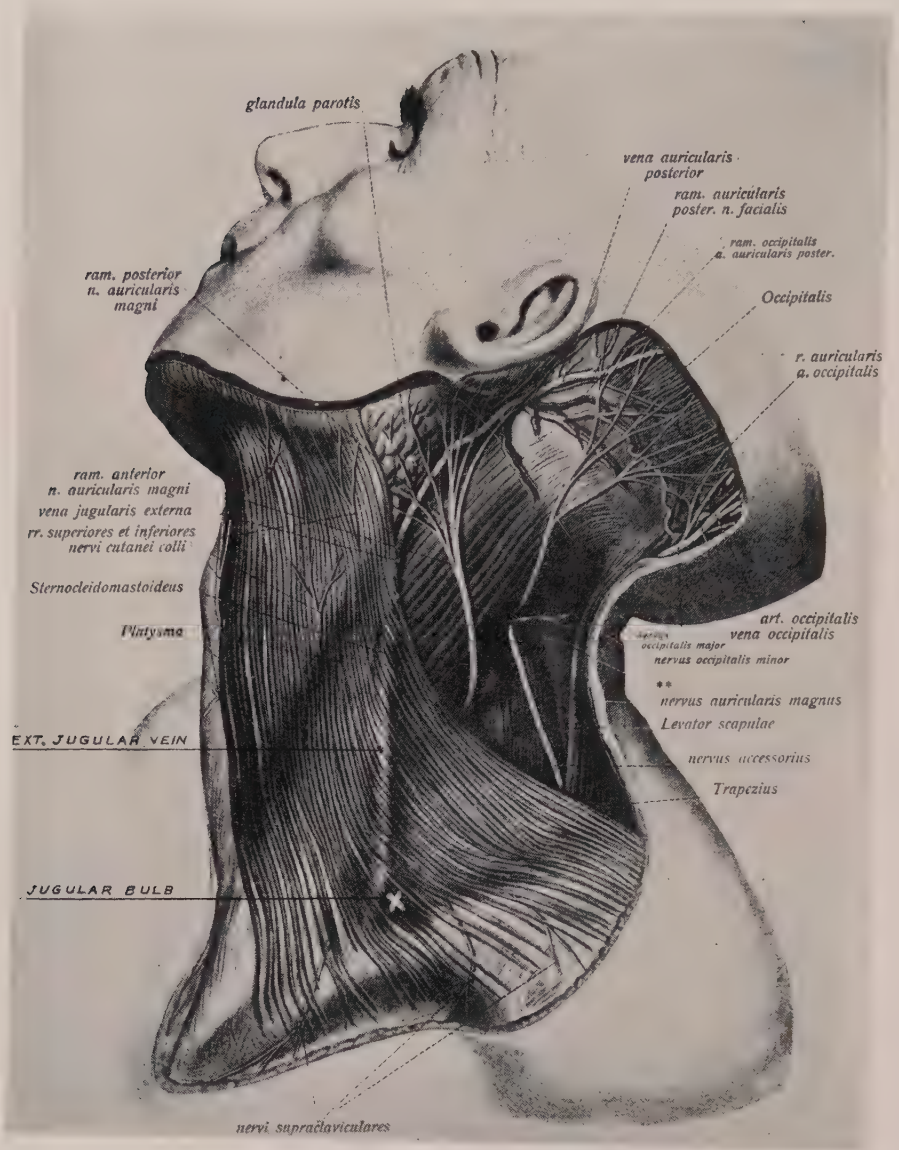

FIG. 18.—Anatomical chart showing the location and general course of the superficial veins in the neck. The external jugular vein is seen beneath the platysma as it crosses the sterno-cleido-mastoid muscle. The jugular bulb, at the root of the vein, is situated in the angle formed by the clavicular insertion of the sterno-cleido-mastoid muscle and the clavicle. (*From Sobotta.*)

advantageous light.[1] If the external jugular is not readily visible, the jugular bulb, situated in the supraclavicular fossa, within the angle formed by the clavicle and the attachment of the sterno-cleido-mastoid muscle, may be visualized. For graphic studies, the bulb is generally serviceable because of its proximity to the heart and consequently because of more marked pressure changes within it. The external jugular vein, because of its superficial situation and length, is often better visualized than the bulb.

In contradistinction to the arterial pulse, which as we have seen is a true propulsion pulse, facilitating the onward flow of blood, the venous pulse is at times referred to as a "stagnation" pulse. The venous blood tends to flow passively into the heart in a steady stream, aided of course, by several outside factors, such as muscle contraction and the aspiratory effect of the thoracic cavity during respiration. The steady flow of venous blood is periodically intercepted and stagnated by the rhythmic contractions of the cardiac chambers, blocking the onflow, as it were. This periodic interception of the venous blood flow manifests itself as a series of volume changes in the veins near the heart. The jugulars, being tributaries of the subclavians which in turn enter into the superior vena cava, are sufficiently near the heart to show these volume changes. Their superficial situation makes them particularly accessible for clinical study.

The mechanism of the venous pulse is best described in conjunction with the study of the components of a graph.

[1] If the patient is reclining and a light is directed toward the head in such a way that the shadow of the clavicle is partly thrown into the supraclavicular fossa, the external jugular vein may generally be visualized. Rhythmic groups of undulations (the jugular pulse) may thus be seen coursing in a cephalad direction. This is best seen at the end of expiration. Such a pulsation imparts practically no impact to the palpating finger.

For details as to the configuration of its component wavelets, graphic methods serve best. The Mackenzie polygraph or the direct optical method with the Frank capsules, described under the study of the arterial pulse and heart sounds, may be used.

In a derived graph (Fig. 19), the following general features are apparent: Wave A is a blunt, upright wave, followed by a depression. It corresponds to auricular contraction which intercepts the venous onflow. This is followed by another upright wave, the C wave. The C wave is somewhat more abrupt in its ascent than the "A" wave, while its descent is sudden and deep. It corresponds to ventricular contraction. Its ascent is due to a sudden rise of intraventricular pressure in early systole, causing a sudden bulging of the auriculo-ventricular valves toward the auricular cavities, giving rise to a sudden increase of pressure within the auricles. This pressure is transmitted by way of the auricular contents to the veins, again intercepting the venous onflow and causing a temporary rise of pressure within them.[1] Its sudden and deep descent is due to the height of ventricular contraction, imparting a pull on the base of the heart and thus suddenly lowering the pressure within the great veins. This phase of the venous pulse is often referred to as the "systolic collapse." It marks the height of systole. During the early part of ventricular relaxation, the auricles fill rapidly and a gradual, passive rise of pressure takes place within the great veins. Upon complete relaxation of the ventricles, when the auriculo-ventricular valves open, the volume of blood accumulated above them suddenly empties into the relaxed

[1] The sudden ascent of the C wave was originally attributed to the carotid artery. There is no doubt that in most venous pulse tracings there is some arterial element. It has been shown, however, that even if the carotid artery is removed, as in experiments, the C component is still present in the venous pulse. Ordinarily, the artery probably merely augments the initial ventricular phase (ascent of C wave) of the venous pulse.

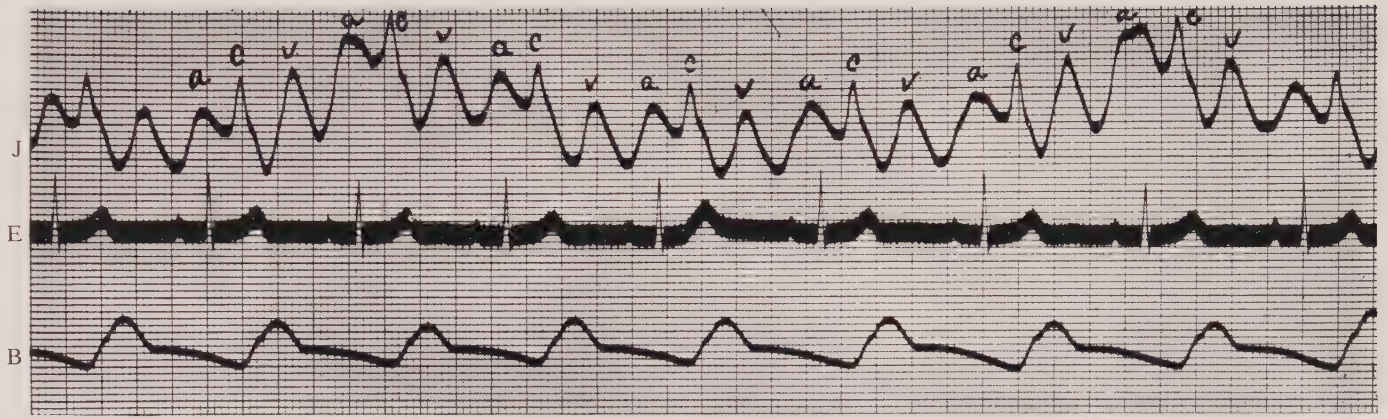

FIG. 19.—Tracing of a jugular pulse (J). It shows the components A, C and V; it also shows the rhythmic rise and fall of the entire pulse wave as a result of pressure variations due to respiration. The synchronous electrocardiogram (E) and brachial pulse (B) serve to indicate the auricular and ventricular phases of the cardiac cycles.

ventricles, causing another and final fall of pressure within the veins. This last gradual rise and the subsequent sudden fall of pressure within the veins is expressed by the v wave in the venous pulse. It is a true diastolic wave. The terminology, a, c and v waves is the one used in most English and American texts; foreign texts often use other letters instead. Additional waves in the curves are also described.

The Venous Pulse as an Index of Intracardiac Events. The venous pulse is a good clinical index of intracardiac events. It records the beginning, duration and termination of auricular and ventricular systoles. *It is the only clinical guide to auricular systole.*

In the clinical study of the cardiac arrhythmias, mere visualization of the venous pulse and the correlation of the time of its appearance with that of the arterial pulse or heart sounds often yield valuable information regarding the nature of certain types of cardiac irregularity.

ARTERIAL AND VENOUS PULSES COMPARED

ARTERIAL	VENOUS
An onward wave, in the direction of the blood flow.	A retrograde wave, against the direction of the blood flow.
The result of a propulsion of the intra-arterial pressure.	The result of interception and stagnation of the venous blood flow.
Detectable in the distal vessels.	Detectable only in the immediate vicinity of the heart.
One single large wave.	Multiple small waves.
Palpable (rarely visible).	Visible (rarely palpable).
Records ventricular systole.	Records auricular and ventricular systole.

Chapter III

The Cardiac Nerves

WHILE the actual production of the cardiac impulse is a function of the specific system, the rate and rhythm with which such impulse is produced, as judged by the rate and rhythm of the corresponding heart muscle response, is known to be influenced by extracardiac factors. It is well known, for instance, that in some individuals the cardiac rate or rhythm, or both, may change very readily and quite perceptibly in response to factors that seem to have their origin outside of the heart. In some individuals, an emotional disturbance or physical pain readily induces alterations in the rate or rhythm of the heart beat; in others, even comparatively mild exaggerations in the normal function of some organ, such as deep breathing or the repeated act of swallowing, may suffice to bring about similar changes.

These observations strongly suggest that the heart is under the influence of some regulatory mechanism, upon the stability of which depends, in part at least, the rhythmic production and the sequential propagation of the cardiac impulse. Clinical and experimental studies point to the *vagus* and *sympathetic* nerves as the chief sources of such regulatory influence.

It is well known that these two sets of nerves are physiological antagonists, the vagi serving as cardiac inhibitors and the sympathetics as cardiac accelerators. Experimental investigations have yielded many interesting facts regarding the function of these nerves and the manner in which they may influence the rate, rhythm and contractility of the heart. The func-

tion of the vagi has been more thoroughly investigated than that of the sympathetics.

The Vagus Nerves

The vagus nerve, especially the right, if stimulated (irritated), exerts a definite inhibitory effect upon the heart beat. It slows the heart action; in addition, it tends to diminish the irritability and the power of contractility of the heart muscle. This latter effect on the heart muscle is evidenced in the auricles only. There seems to be no such influence exerted on the ventricles, due probably, as some contend, to their lower gradient of vagus supply. However, it is not unreasonable to suppose that, even if such influence were exerted upon the ventricular musculature, it would probably be, at least partly, compensated by the augmenting effect of a more perfect ventricular filling during the long diastoles incident to vagus inhibition.[1] Marked vagus slowing of the heart is associated with a fall in blood pressure.

Clinically, in the human subject, slowing of the heart can often be induced by firm pressure over the carotid sheath in the neck carrying the vagus nerve, or by pressure over the eyeballs.[2] Pressure over the right carotid is generally more effectual. In susceptible individuals, or those under the influ-

[1] A long diastole incident to vagus inhibition permits greater filling of the cardiac chambers; as a result, the initial tension on the ventricular muscle fibers is greater and the resulting muscular contraction is more powerful. The actual output per beat is therefore increased when the heart is slowed.

[2] What we clinically term vagus pressure is in reality only a reflex vagus slowing. The vagus nerve in the neck is situated too deep to permit of actual pressure upon it; what we really press is the carotid artery. The vagus slowing is probably a reflex phenomenon by way of the vagus nucleus. The frequent vagus response to eyeball pressure tends to confirm this. In susceptible individuals vagus slowing can be induced by irritating any peripheral nerve.

ence of vagotropic drugs, "vagus pressure" is not without some element of discomfort or even risk. The cardiac slowing thus induced may be profound. Individuals who are naturally vagus-sensitive, or those sensitized by vagotropic drugs or toxins, often react to a rather marked degree, even to apparently trivial causes. In such individuals a sudden tap over the abdomen, the sudden inflicting of pain or an unpleasant scene —a psychic shock, such as the sight of flowing blood, especially their own blood—may induce marked transitory slowing of the heart often associated with giddiness or fainting.

In addition to the inhibitory influence of the vagi on the rate, rhythm, muscle irritability and contractility of the heart, physiological observations suggest that these nerves also have a tendency to a selective, regional distribution. The right vagus is generally distributed to the region of the cardiac pacemaker and the left vagus generally supplies the region of the junctional tissues and the ventricles. Stimulation of the *right vagus* slows the heart by depressing the rate of impulse production at the cardiac pacemaker. In such a case the slowing effect is primarily exerted upon the auricles which in turn set a correspondingly slow rate for the ventricles. That the right vagus is distributed mainly to the region of the cardiac pacemaker is further suggested by the observation that if in an experimental animal the sino-auricular node is destroyed or depressed, stimulation of the right vagus nerve fails to slow the heart.[1] *Left vagus* stimulation, on the other hand, tends to slow the heart principally by depressing impulse conduction in the region of the junction tissues (A-V node and bundle). In such a case, because of interference with the transmission of the impulse to the ven-

[1] Cohn, A. E. The effect of morphine on the mechanism of the dog's heart after removal of one vagus nerve. *J. Exper. M.*, 1913, xviii, 715.

tricles, the ventricular rate may be slow and may be independent of the auricular rate.

It must, of course, be understood that the regional distribution of these nerves is not absolute, and that there are many variations encountered, both clinically and experimentally.

The Sympathetic Nerves

Our knowledge of the detailed function of the sympathetic nerves is not nearly as complete as that concerning the vagi. This is largely due to the anatomical situation of these nerves, which renders them comparatively inaccessible for detailed investigation.

In animals, the anatomical "relay" from which issue the postganglionic fibers of the cardiac sympathetic nerves is situated within the thoracic cavity: it is the "stellate ganglion," the first thoracic sympathetic ganglion. In man, the paths of the sympathetic fibers going to the heart are seemingly more complex. Their course, at any rate, is not as yet sufficiently understood to permit of detailed description. The fibers are known to arise in the upper thoracic spinal cord (levels I to IV) and to enter corresponding sympathetic ganglia, where they are relayed. The postganglionic fibers going to the cardiac plexuses probably issue by way of the inferior and middle cervical sympathetic ganglia.

Such anatomical relations of the sympathetic cardiac nerves, particularly in the human subject preclude the possibility of a detailed functional study.

In the experimental animal, stimulation of the *right stellate ganglion* is known to accelerate both the auricles and the ventricles. During such acceleration graphic records show that the cardiac impulse arises at the normal site of the cardiac pace-

maker and that it traverses its usual path. Stimulation of the *left stellate ganglion* accelerates the heart similarly, but graphic registration in these cases occasionally reveals an added phenomenon, namely, that the auricles and ventricles at times beat almost synchronously, or that in some instances contraction of the ventricles actually precedes that of the auricles, indicating that the pacemaker has apparently been shifted to a lower level. In such cases, instead of the S-A node, which normally gives rise to a downward spread of the cardiac impulse, it is the A-V node that generally sets the pace, causing the impulse to spread from its new point of origin in two opposite directions, upward to the auricles and downward to the ventricles. Thus the effect of left sympathetic stimulation is shown to be manifested largely in the region of the junctional tissues.

Summary

In summary, it may be said that the cardiac nerves, vagus and sympathetic, are physiological antagonists, the vagi serving as inhibitors of rate, irritability and contractility, and the sympathetics acting as accelerators and augmentors. It may also be said that these nerves have a tendency to a selective regional distribution; the right nerves being generally distributed to the region of the S-A node, the cardiac pacemaker, while the left nerves supply mainly the region of the A-V node and ventricles.

These two sets of physiological antagonists exert their respective influences upon the heart constantly, one tending to retard and the other to accelerate it. The rate and at times the rhythm of the heart beat therefore may be regarded as expressing the resultant of their combined effects.

It becomes evident that a proper stable balance between these two sets of nerves is essential to the maintenance of a normal cardiac rhythm; and conversely, that a disturbance in the physiological balance between these two sets of antagonists may account for some of the forms of cardiac arrhythmia.

Chapter IV

The Diagrammatic Representation of the Intrinsic Mechanism and Extracardiac Manifestations of the Heart Beat

In order to facilitate the understanding of the intrinsic mechanism of the normal heart beat as well as that of its peripheral manifestations, the attempt is made to represent them in diagrammatic form, in which the various phases of the heart beat are correlated with corresponding phases of its extracardiac features. They are represented in the order and time of their relative appearance; the approximate time of duration of each of their respective components, in the course of a normal cardiac cycle is also designated.

In these diagrams, the conventional use of lines and blocks, to depict the intrinsic cardiac mechanism, is employed; they are further elaborated, however, to designate the approximate duration of the various phases of the cardiac cycle, such as heart muscle response, and the various intervals that elapse in the course of the sequential propagation of the cardiac impulse. The electrical phenomena, heart sounds, venous and arterial pulses are all drawn to scale in order that the relative appearance of their respective components in a cardiac cycle may be viewed by comparison and that therefore their interrelation may be more readily appreciated.

Such diagrammatic correlation of the various phenomena incident to the heart beat will enable us, it is hoped, to understand more clearly not only the mechanism of the normal heart beat, but also those aberrations from the normal which may constitute some of the cardiac arrhythmias.

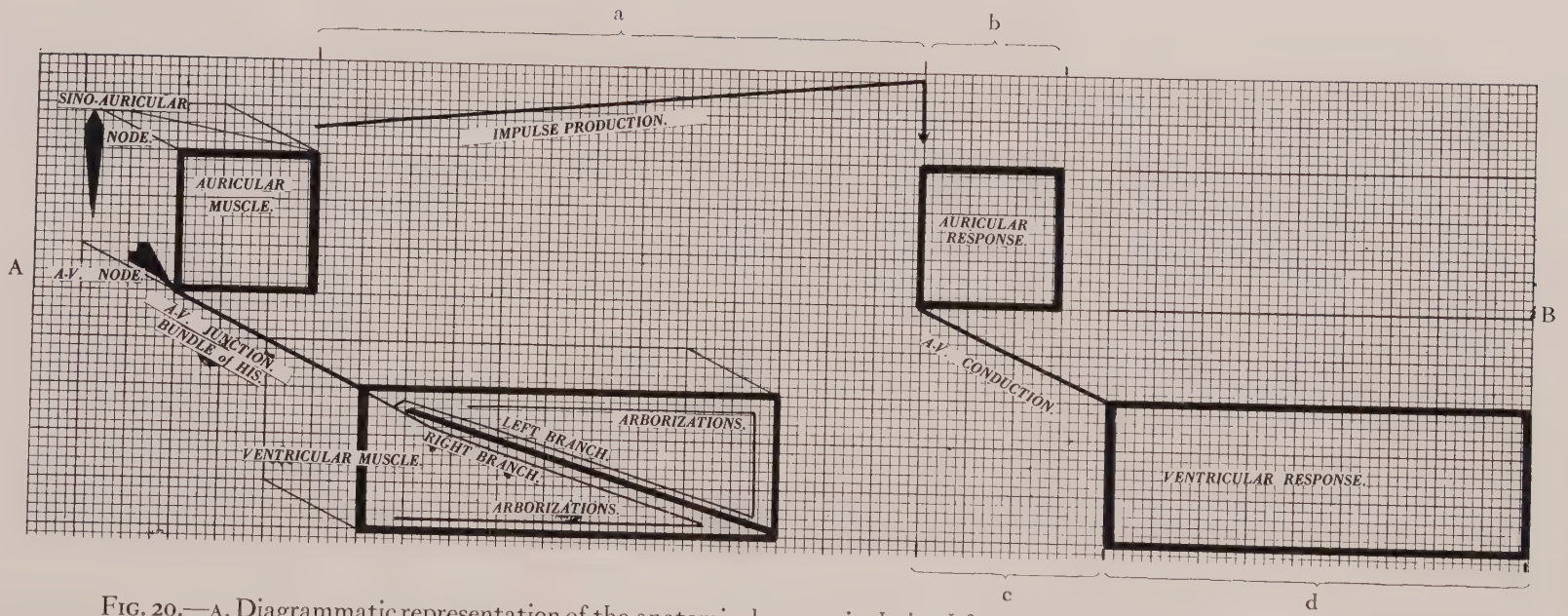

FIG. 20.—A. Diagrammatic representation of the anatomical levels within the heart. It shows, from above downward, the following zones: the auricles containing the S-A and the A-V nodes; the A-V junction with the bundle of His: and the ventricles containing the two main branches and the terminal arborizations of the bundle.

B. is derived from A and represents, from left to right, the physiological sequence of events within the heart. It shows the approximate duration of impulse production (a), auricular response (b), A-V conduction time (c) and ventricular response (d). Time: Vertical heavy lines = $\frac{1}{25}$ second.

Figure 20 is presented in order to introduce the diagrammatic form to be employed. It represents, from above downward, the anatomical zones within the heart (A), and, from left to right, the physiological sequence of events that transpire during a normal cardiac cycle (B).

Figure 21 shows the appearance of the various components of the extracardiac manifestations of the heart beat (E.C.G., heart sounds, arterial and venous pulses) in relation to each other, and in relation to corresponding phases of the intrinsic cardiac mechanism.

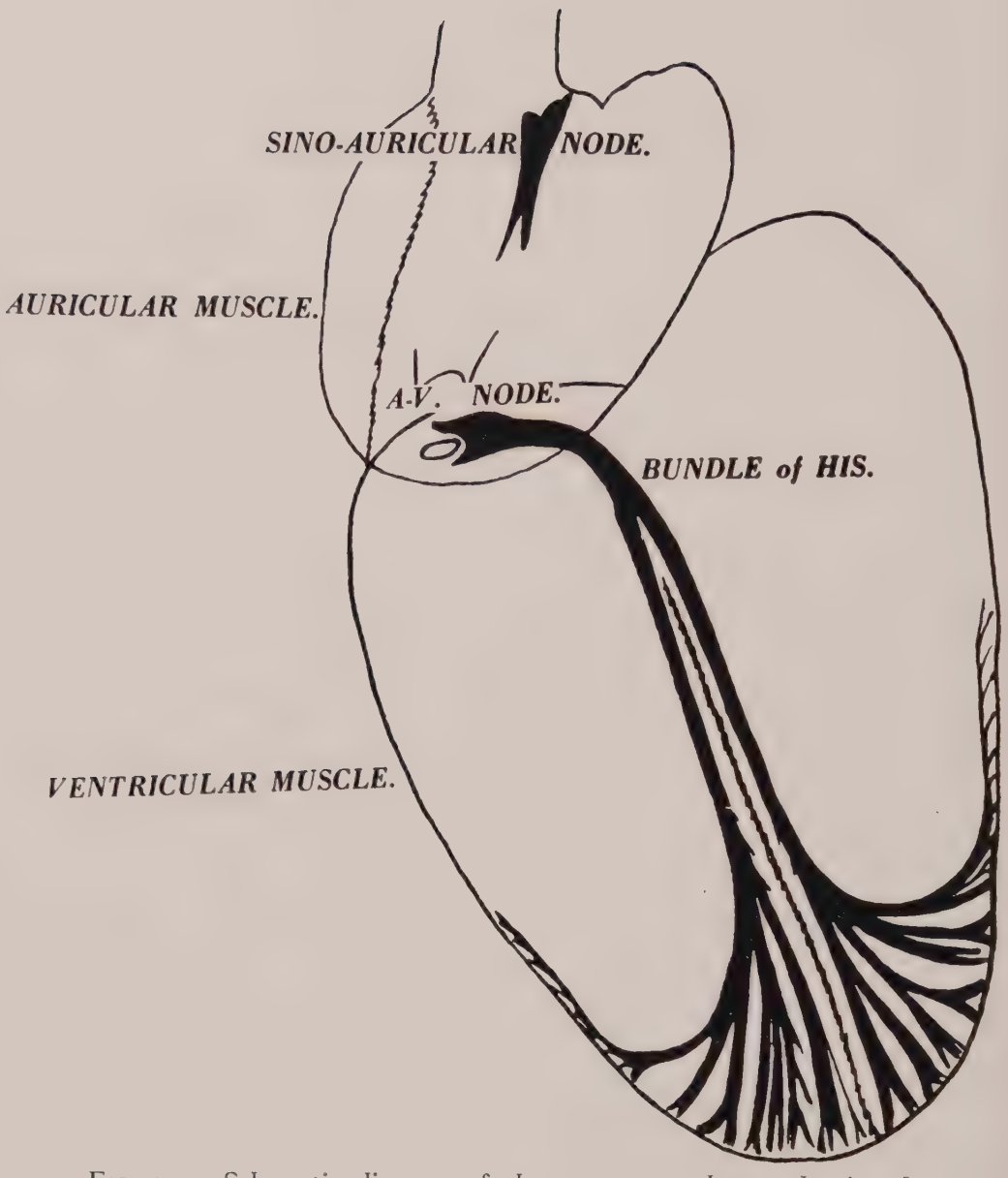

FIG. 21A.—Schematic diagram of the transparent heart, showing the specific system.

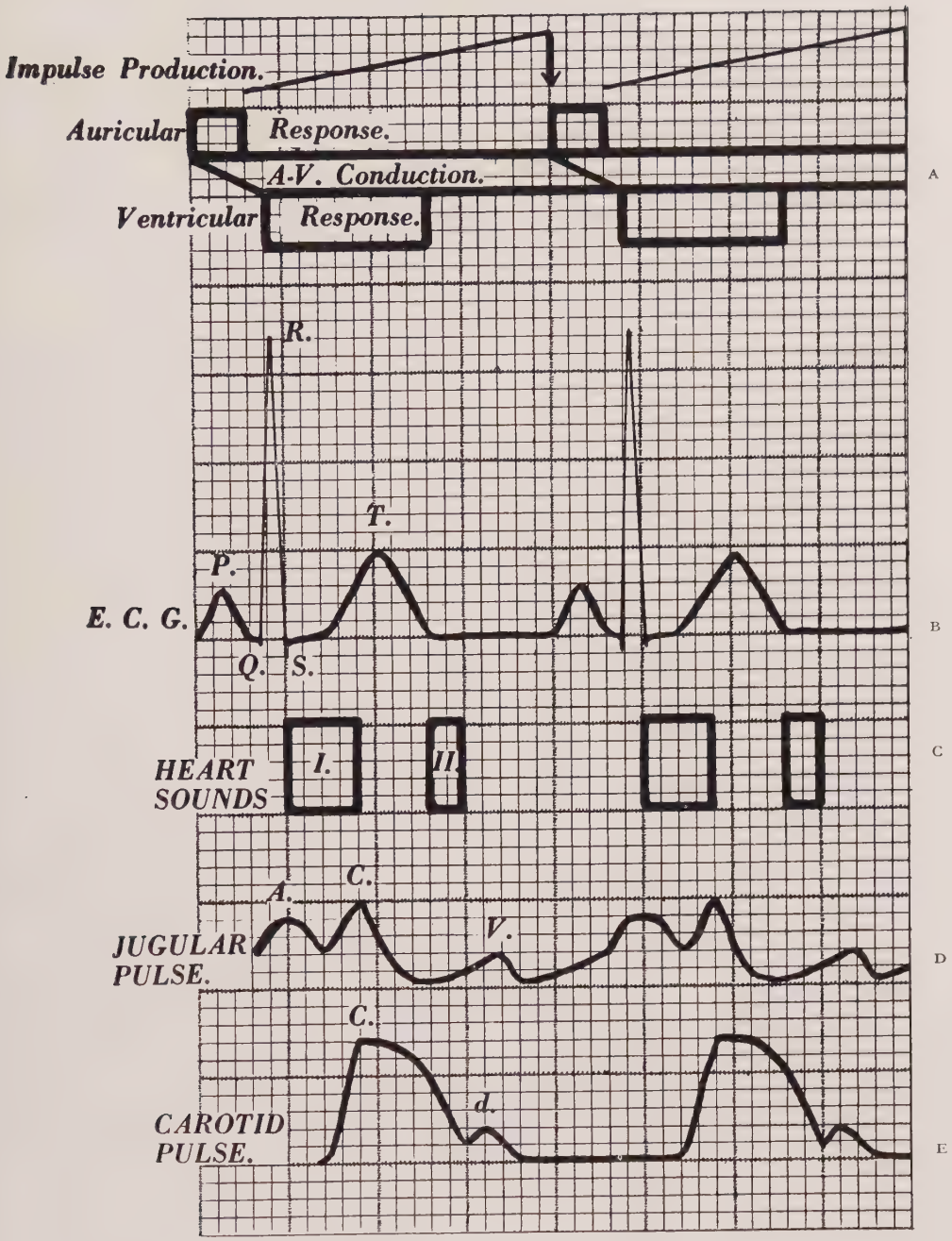

FIG. 21B.—Diagrammatic representation of the intrinsic cardiac mechanism (A) correlated with the various extracardiac manifestations of the heart beat; electrical changes, E. C. G. (B), heart sound (C), venous pulse (D) and arterial pulse (E).

Chapter V

Essential Characteristics of the Normal Rhythmic Heart

THE clinically normal rhythmic heart presupposes a rhythmic recurrence of similar intracardiac events from cycle to cycle as well as a correspondingly rhythmic recurrence of the various components of the extracardiac manifestations of the heart beat. The anatomical and physiological integrity of the specific system and a moderately stable balance of the antagonistic members of the cardiac regulatory mechanism, vagus and sympathetic, are implied. Figure 22 represents such a normal rhythmic heart beat.

It shows that the cardiac impulse is rhythmically built up at the site of the normal cardiac pacemaker and that it is transmitted along the usual pathways of conduction, in a rhythmic and sequential manner. The time required for impulse production and for the transmission of the impulse over any given part of its path is constant from cycle to cycle. It also shows a correspondingly rhythmic reappearance of all the extracardiac manifestations of the heart beat: electrical manifestations, heart sounds, arterial and venous pulses. It shows, furthermore, that the general characteristics of these extracardiac features are maintained throughout from beat to beat. If the diagram is viewed as a symbolical representative of clinically ascertainable phenomena, it may be interpreted to represent not merely a group of graphic records but rather a group of clinical features of the heart beat, each of which normally presents certain characteristic clinical aspects. For instance in the electrocardiographic curve (E.C.G.) we see a

rhythmic recurrence of similar groups of characteristic complexes separated by similar isoelectric intervals. Similarly the heart sounds, first and second, "ring true" from beat to beat and their pitch and duration do not vary to any perceptible degree. Also the arterial pulse reappears under the palpating finger with a regularity of rhythm and a constancy of size. The jugular pulse (less constant because of respiratory variations in intrathoracic pressure) as well presents rhythmically recurring groups of fine waves, most apparent to the observer at the end of expiration.

In graphic records, the intervals designating conduction time, such as the P-R interval in the electrocardiogram and the A-C interval in the venous curve, when measured, show a constantly equal time relation for corresponding phases, in all cycles.

The Degree of Departure from Normal Rhythm as a Guide to the Degree of Functional or Anatomical Impairment of the Heart

A disturbance in the rhythmic production of the cardiac impulse, or a disturbance in its transmission along the usual channels, generally manifests itself as a cardiac arrhythmia. The degree or type of the irregularity, however, is not necessarily a reliable guide to the degree of functional disturbance in the heart. A slight disturbance in function may yield no irregularity at all and may become evident only with graphic aid. Likewise, a markedly advanced functional or anatomical disturbance may manifest itself only by an unusually rapid or an unusually slow heart rate, unaccompanied by arrhythmia.

Extremes in functional impairment, therefore, may at times yield similar clinical pictures, as far as the rate and

FIG. 22.—Diagrammatic representation of the mechanism and extracardiac manifestations of the Normal Rhythmic Heart.

A, *Mechanism:* Rhythmic production of the cardiac impulse; rhythmic auricular responses; A-V conduction time = 0.16 seconds. Rhythmic ventricular responses.

B, *E. C. G.:* Groups of normal complexes (P,Q,R,S,T) recurring rhythmically. P-R interval = 0.16 seconds; QRS interval = 0.04 seconds.

C, *Heart Sounds:* First and second heart sounds show a constancy in duration and intensity from beat to beat.

D, *Jugular Pulse:* Rhythmically recurring groups of waves corresponding to the auricular (A), ventricular (C) and diastolic (V) volume changes in the venous pulse.

E, *Carotid Pulse:* Equally spaced arterial pulse waves showing uniform pulse crests (c) and dicrotic notches (d).

(In this and in all subsequent similar diagrams, vertical lines in the background designate time; heavy lines. = 1/5 second); fine lines = 1/25 second. The horizontal lines indicate the amplitude of the various waves in millimeters.

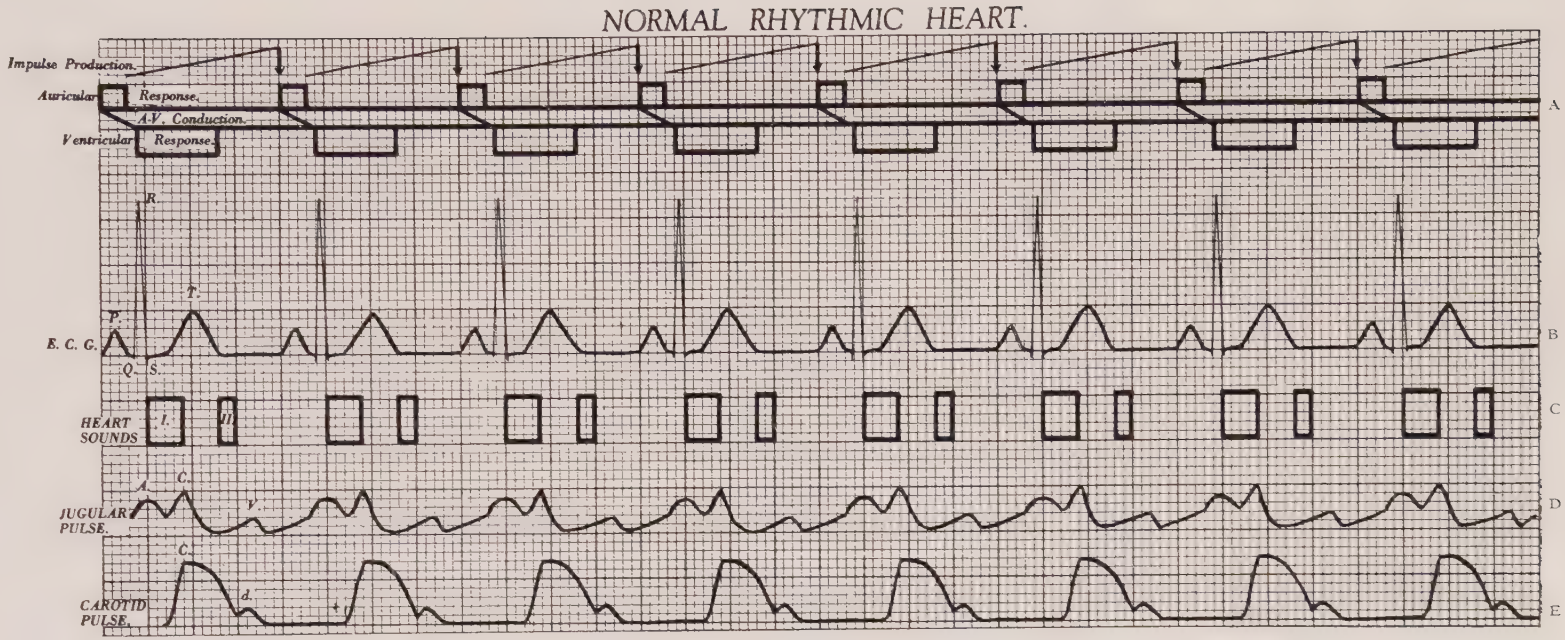
NORMAL RHYTHMIC HEART.
Impulse Production.
Auricular
Response.
A.V. Conduction.
Ventricular
Response.
A
R.
T.
P.
E. C. G.
Q.
S.
B
HEART SOUNDS
I.
II.
C
A.
C.
JUGULAR PULSE.
V.
D
C.
CAROTID PULSE.
d.
E

FIG. 22.

rhythm of the heart are concerned. Irregular heart action, on the other hand, may at times accompany any degree of functional or anatomical disorder. It is therefore merely an expression of the type but not necessarily of the degree of the impairment. Because of this, it is best to evaluate the clinical significance of an irregular heart beat in the light of all other available clinical data, in any given cardiac disorder.

PART TWO
THE CARDIAC ARRHYTHMIAS

Introduction to Part Two

THE clinical irregularities of the heart beat may be conveniently classified into five general groups:

1. Sinus Arrhythmias. These comprise a group of disorders in which the disturbance in function arises primarily at or near the cardiac pacemaker, the S-A node.

2. Auriculo-ventricular Heart Block. Disturbance in mechanism in this group is produced by some interference with the transmission of the cardiac impulse across the A-V junction (A-V node and bundle of His).

3. Extrasystolic Arrhythmias. In these a subsidiary center occasionally succeeds in elaborating and discharging a premature cardiac impulse, or a series of such impulses, which, because of their untimely appearance, anticipate the normal rhythmic sinus impulse. They disturb the rhythm of the heart beat by inducing an occasional premature cardiac contraction, of auricles or ventricles or both.

If the premature beats occur successively and in large numbers, instead of inducing an irregularity, they produce a paroxysmal alteration of the heart rate which may last a varying length of time. Such periods of rapid heart action constitute the *paroxysmal tachycardias.*

4. Auricular Flutter and Auricular Fibrillation. In these the cardiac pacemaker is apparently silent and the auricular musculature is activated by a self-perpetuating stimulus traveling rapidly in a closed and circuitous path.[1] The ventric-

[1] The term circus or circular path is generally used to designate the path of the impulse within the auricles in flutter or fibrillation. Since such a path, however, is anything but a circle, but may instead be any ill-defined, roundabout, circular, oval or any other form of a *closed path*, the term circuitous is employed throughout the text dealing with these disorders.

ular rate depends largely upon the relative physiological integrity of the A-V junctional tissues.

5. Combined Arrhythmias. As the name implies, these are cases in which two or more of the above-mentioned arrhythmias coexist. A combination of two disorders may at times render the heart beat regular.

Chapter VI

Sinus Arrhythmias

THE sinus arrhythmias comprise a group of cardiac irregularities in which the disturbance of rhythm is a direct result of some physiological or anatomical impairment at or near the site of the cardiac pacemaker, the sino-auricular node, hence the name, "sinus arrhythmias." Because of some alteration in its rhythmic metabolic function, the pacemaker at times fails to produce the cardiac impulse in the usual rhythmic manner. Consequently, it *sets an irregular pace* which the whole heart follows with a correspondingly irregular response.

The most potent factor initiating such disturbance in the rhythmic production and discharge of the cardiac stimulus is a disturbed balance of power on the part of the two antagonistic cardiac regulatory nerves, the vagus and sympathetic. In order to appreciate the clinical features of the sinus arrhythmias, let it be recalled that the normal rhythmic heart beat presupposes not only the integrity of the ordinary heart muscle and that of the specific system, but also a normal, stable balance between the antagonistic, vagus and sympathetic, nerves.

The ideal rhythmic heart *rate* of 72 is not the product of the inherent cardiac automatism. Such rate, on ultimate analysis, is but a resultant of two opposing forces, the sympathetic accelerators and the vagus inhibitors. Normally, these two nerves constantly influence the pacemaker, the former activating it and the latter holding it in leash. Experimental blocking of the vagus, especially the right, or the clinical removal of its influence by means of atropine, approximately doubles the

heart rate, showing the profound restraining influence of the vagi on the heart, even under normal conditions.

In some individuals, a high vagus tone or an unstable vagus sympathetic balance is inherent. In others, it is readily induced by extraneous factors. Vagotropic toxins, such as those present in cholemia, uremia or pregnancy, or vagotropic drugs, such as digitalis, pilocarpine or morphine, may serve to induce a heightened vagus tone.

Under such circumstances, the vagus curbs the cardiac pacemaker excessively, and its influence may manifest itself clinically as an alteration in the rate or the rhythm of the heart beat. If the inhibition of the cardiac pacemaker is constant, a relatively uniform, slow heart rate results. If inconstant, an arrhythmia supervenes in which the slow phases of the irregularity correspond to the temporary rise in vagus tone. Clinically, the slow phases are characterized by a complete standstill of the whole heart.

Types of Sinus Arrhythmia

1. respiratory sinus arrhythmia (fig. 23)

This type is the most common form. It is characterized by a rhythmic waxing and waning of the heart rate. Groups of slower cardiac cycles alternate with groups of more rapid cycles and the *alternations correspond to the variations in the respiratory phases of the subject.* Such hearts accelerate during inspiration and slow during expiration: at times, the irregularity is inconspicuous and is brought forth only on forced breathing; while at other times, the expiratory slowing is very marked. In children, in whom the respiration may be very irregular, as during sleep or during crying spells, the arrhythmia may at

times seem totally irregular and therefore quite puzzling to those unaware of such possible variation from the usual type.

Respiratory sinus arrhythmia is the most common form of arrhythmia in young children and in those convalescing from acute diseases. It has no special clinical significance. It does not denote disease.

2. "PHASIC" SINUS ARRHYTHMIA (FIG. 24)

This type is comparatively rare. It is characterized by a periodic retardation of the whole heart for 6 to 12 beats, followed by a gradual acceleration. The slowing may occur regularly or it may appear only from time to time. This arrhythmia *bears no relation to respiration.* It is at times seen in the course of prolonged digitalis medication. Such patients may exhibit phasic sinus slowing during the early periods of rest following exercise.

3. GRADUAL AND PROFOUND SLOWING OF THE WHOLE HEART

Some individuals are subject to occasional profound slowing of the heart in response to apparently trivial causes, among which psychic factors predominate. In such individuals, the sight of blood, or a horrifying or disgusting scene, may induce a train of well-marked clinical symptoms such as a sense of weakness, giddiness and fainting, together with pallor, sweating, fall of blood pressure and a gradual, profound slowing of the heart.[1] Such symptoms and signs are most likely due to a marked temporary increase in vagus tone in response to a

[1] Cotton and Lewis report some interesting observations on soldiers subject to fainting spells at the sight of blood (Wassermann tests). In one case, anticipating the attack, close observations were made and the following noted: At the sight of blood, patient became pale, was covered with cold sweat, the blood pressure fell and the heart rate slowed. When the heart rate fell to about 45, patient fainted. Intravenous atropine (grain $\frac{1}{30}$) brought about prompt recovery. (Cotton, T. F., and Lewis, T. Observations upon fainting attacks due to inhibitory cardiac impulses. *Heart*, Lond., 1918, viii, 23–24.)

FIG. 23.—Diagrammatic representation of the mechanism and extracardiac manifestations of Sinus Arrhythmia (Respiratory).

A, *Mechanism:* Groups of cardiac cycles, in which impulse production is delayed, alternate with groups in which impulse production is accelerated. Every impulse, when discharged, is followed by a normal sequence of events.

B, *E. C. G.:* Groups of slow cardiac cycles alternate with groups occurring in rapid succession. Individual complexes (P, Q, R, S, T) are normal.

C, *Heart Sounds:* Groups of slowly appearing heart sounds alternate with groups occurring at a more rapid rate.

D, *Jugular Pulse:* Groups of slower venous pulse beats alternate with groups more closely spaced. The forms of the individual waves are unaltered.

E, *Carotid Pulse:* "A waxing and waning of the pulse beat." Groups of slow pulse beats alternate with groups of quickened pulse beats.

(The heart rate is accelerated during inspiration and retarded during expiration.)

SINUS ARRHYTHMIA (RESPIRATORY)

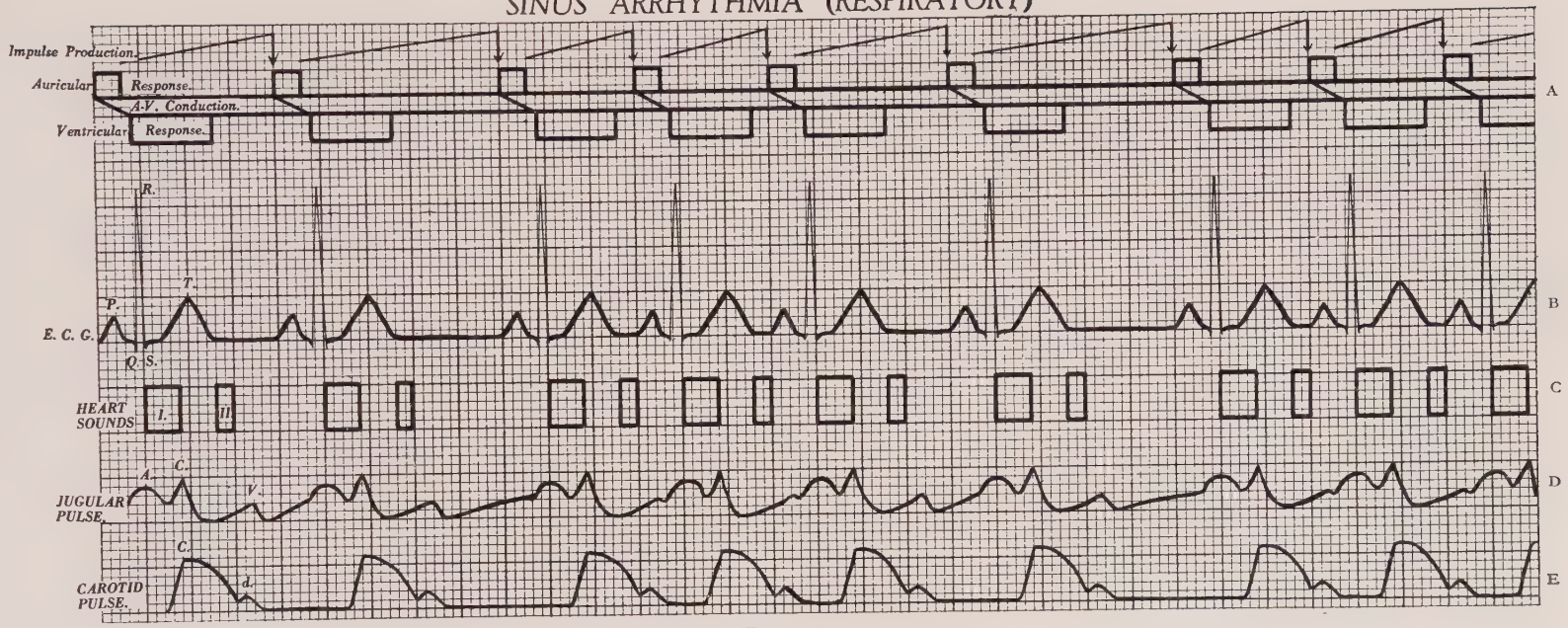

FIG. 23.

FIG. 24.—Diagrammatic representation of the mechanism and extracardiac manifestations of Phasic Sinus Slowing.

A, *Mechanism:* Occasional period in which impulse production is unusually retarded for a few beats. (One such period is represented, beginning with cycle 2 and ending with cycle 5.) If impulse production is markedly delayed, a secondary center may initiate an impulse as shown in cycle 5. Such an impulse in its spread may destroy the slowly forming immature sinus impulse as indicated by crossing of broken lines in the zone of impulse production in cycle 5.

B, *E. C. G.:* Occasional groups of cycles in which the diastolic period is gradually prolonged from beat to beat. Individual complexes are normal. Rarely, after a very long diastole the auricular P wave may be distorted (inverted) indicating the "escape of a beat" from an ectopic focus. Cycle 5 shows such an "escaped nodal beat."

C, *Heart Sounds:* Periodic gradual delay in the appearance of the heart sounds. In cycle 5 the heart sounds are loudest because they follow in the wake of a long pause.

D, *Jugular Pulse:* Periodic retardation of the venous pulse from cycle to cycle.

E, *Carotid Pulse:* Marked slowing of the arterial pulse for a few beats. In cycle 5 the pulse beat following the long pause is largest.

(In this type of disorder the gradual retardation of the heart beat bears *no relation to respiration.*)

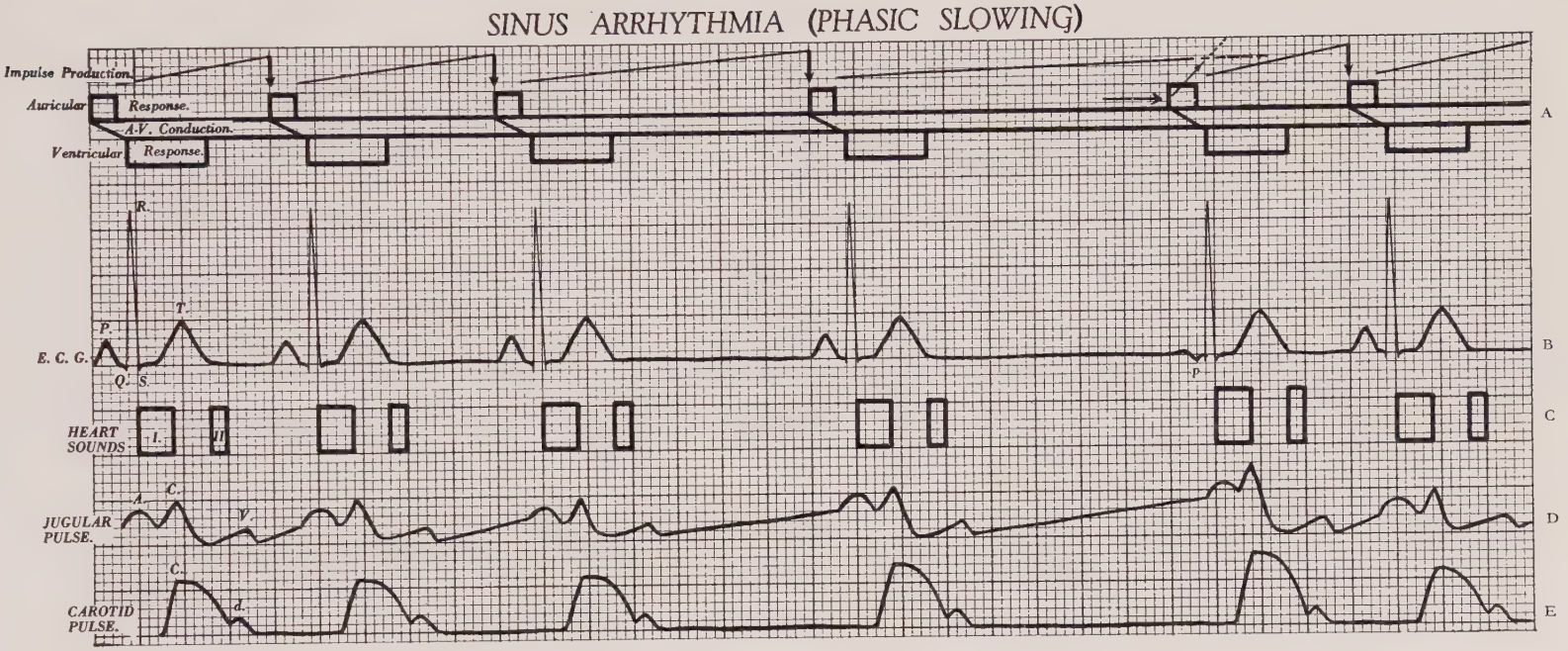
SINUS ARRHYTHMIA (PHASIC SLOWING)
Impulse Production.
Auricular
Response.
A-V. Conduction.
Ventricular
Response.
A
R.
T.
P.
E. C. G.
Q.
S.
P.
B
HEART SOUNDS
I.
II.
C
A.
C.
V.
JUGULAR PULSE.
D
C.
d.
CAROTID PULSE.
E

FIG. 24.

psychic stimulus causing, for the time being, a profound depression of the cardiac pacemaker.

Clinically, marked slowing of the heart may at times be induced by pressure over the carotid sheath in the neck (especially over the right) or by pressure over the eyeballs.

During the long diastoles, incident to the marked slowing, the entire heart is at a standstill, as evidenced by the fact that heart sounds, arterial pulse and jugular pulse are all silent. In the electrocardiogram, except for long diastoles, there is no notable alteration in form of the individual complexes. At times, if the slowing is profound and the corresponding diastole very long, the normal pacemaker being depressed and therefore silent, an ectopic impulse from a secondary center may take up its function temporarily and initiate a heart beat (Fig. 24, Cycle 5).

4. "SINO-AURICULAR BLOCK" (FIG. 25)

This condition is characterized clinically by an occasional "dropping" of a heart beat in the course of an apparently rhythmic heart action, leading to an occasional pause or slow phase and consequently to an occasional break in the rhythm of the heart beat. The resulting slow phases correspond approximately to the length of two normal cardiac cycles. During the long pauses, clinical observations and graphic records show that the entire heart is at a standstill, indicating that the source of the disturbance is at or near the cardiac pacemaker. Exercise tends to eliminate the arrhythmia.

The underlying mechanism of this disturbance is not fully understood; at any rate, there is no common agreement as to the nature of it. Some regard it as a temporary standstill of the

cardiac pacemaker; namely, that the S-A node occasionally fails to produce a stimulus. Others are inclined to the belief that the pacemaker does elaborate successive stimuli in the usual rhythmic fashion, but that occasionally a stimulus fails to invade the auricular musculature. This latter theory assumes that there is an impediment to the spread of the stimulus at the sino-auricular junction; namely, that there is a block. For this reason the name, "sino-auricular block," has been adopted. Those interested in the relative merits of the two conflicting theories are referred to the literature on the subject, of which there is an abundance.[1]

It is of clinical interest that this so-called "sino-auricular block," which is essentially an arrhythmia, may mask itself as a simple bradycardia (Fig. 26). In such a case, if we accept the theory of "block," we may assume that every alternate sinus impulse fails to induce an auricular response. The true nature of such clinical disorder may be recognized by the fact that on exercise, instead of a gradual acceleration, as occurs in simple bradycardias, such slow heart *suddenly doubles its rate.* If further exercise is carried on, the subsequent acceleration is gradual. On rest, following the exercise, there is a slight gradual retardation, after which the rate may suddenly fall to one-half, that is, to the rate of the original bradycardia. Post-infectious bradycardias or the bradycardia of athletes with rates of about 40 a minute ought to be investigated, by determining the type of their reaction to exercise, in order that

[1] References on the nature of "sino-auricular block":

Wenckebach, K. F. Beiträge zur Kenntnis der menschlichen Herztätigkeit. *Archiv. f. Anat. u. Physiol.* (physiol. Abt.), 1906, pp. 297–354; 1907, pp. 1–24; 1908, pp. 53–86.

Lewis, T. The Mechanism and Graphic Registration of the Heart Beat. Ed. 3, Lond., 1925, p. 411.

Resnik, W. H. Nature of so-called sino-auricular block. *Arch. Int. Med.*, 1925, xxxvi, 788.

FIG. 25.—Diagrammatic representation of the mechanism and extracardiac manifestations of "Sino-Auricular Block." (Occasional 2:1 block.)[1]

A, *Mechanism:* Rhythmic production of cardiac impulse. An occasional impulse fails to induce an auricular response and therefore a complete cardiac cycle is now and then lost.[2]

B, *E. C. G.:* Occasional long diastole corresponding approximately to two normal ones.

C, *Heart Sounds:* Occasional absence of a set of (first and second) heart sounds. Heart sounds following the pauses are louder.

D, *Jugular Pulse:* Occasional pause in the venous pulse approximately equal to the length of two normal cycles.

E, *Carotid Pulse:* Occasional pause in the arterial pulse approximately equal to two normal pulse intervals. The pulse following a pause is large.

[1]The term 2:1 block in this case implies a ratio between the frequency of impulse production and the auricular response.

[2]The mechanism herein described is the one generally accepted. For further discussion see p. 76.

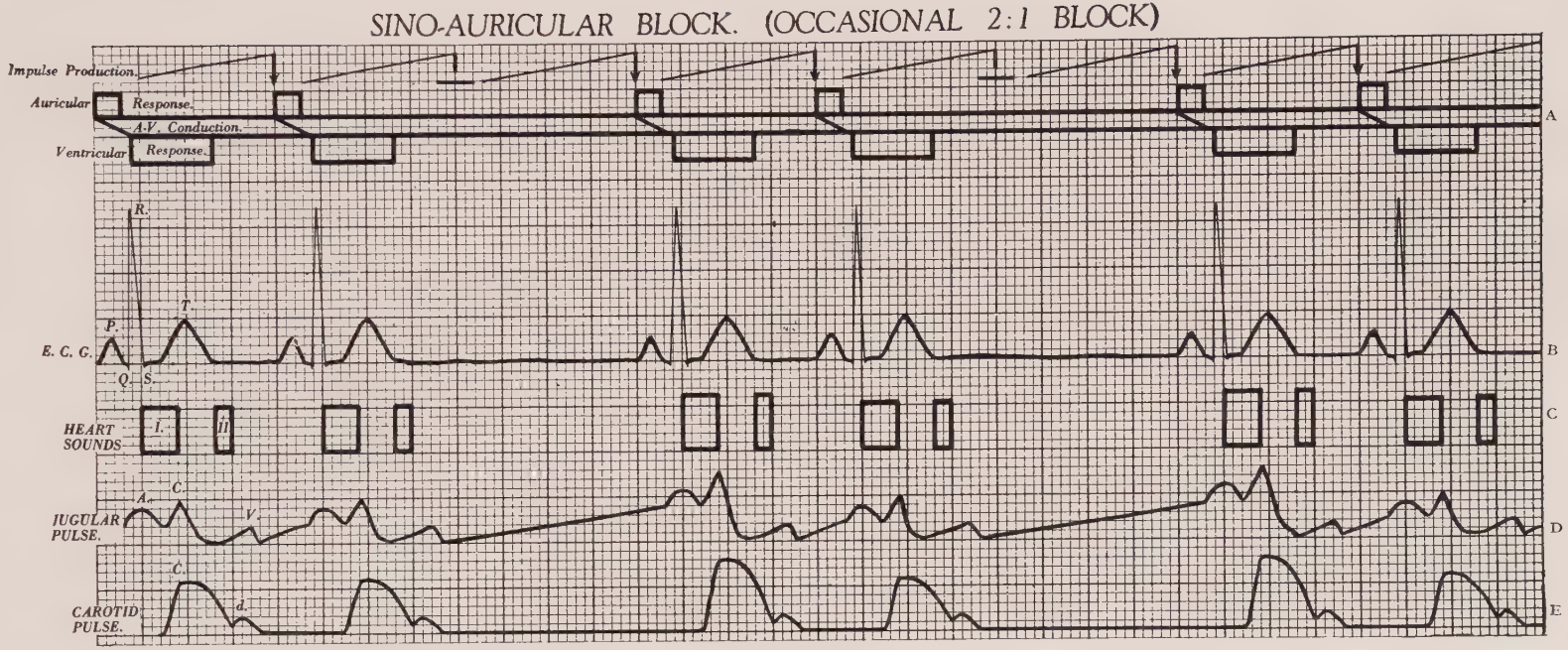
SINO-AURICULAR BLOCK. (OCCASIONAL 2:1 BLOCK)
Impulse Production.
Auricular
Response.
A-V. Conduction.
Ventricular
Response.
A
R.
P.
T
E. C. G.
Q.
S.
B
HEART SOUNDS
I.
II
C
A.
C.
V.
JUGULAR PULSE.
D
C.
d.
CAROTID PULSE.
E

FIG. 25.

FIG. 26.—Diagrammatic representation of the mechanism and extracardiac manifestations of "Sino-Auricular Block" (Persistent 2:1 block).

A, *Mechanism:* Rhythmic production of cardiac impulse: alternate impulses fail to activate the auricles; therefore, every alternate complete cardiac cycle is missing.

B, *E. C. G.:* Simple bradycardia with normal complexes.

C, *Heart Sounds:* Slowly appearing, equally spaced heart sounds.

D, *Jugular Pulse:* Slowly appearing, equally spaced venous pulse groups.

E, *Carotid Pulse:* Equally spaced, slow arterial pulse beats.

(The "block" theory assumes that each alternate impulse is "blocked" at the sino-auricular junction. The opposing theory supposes that there is a periodic standstill of the pacemaker. See text, p. 77.)

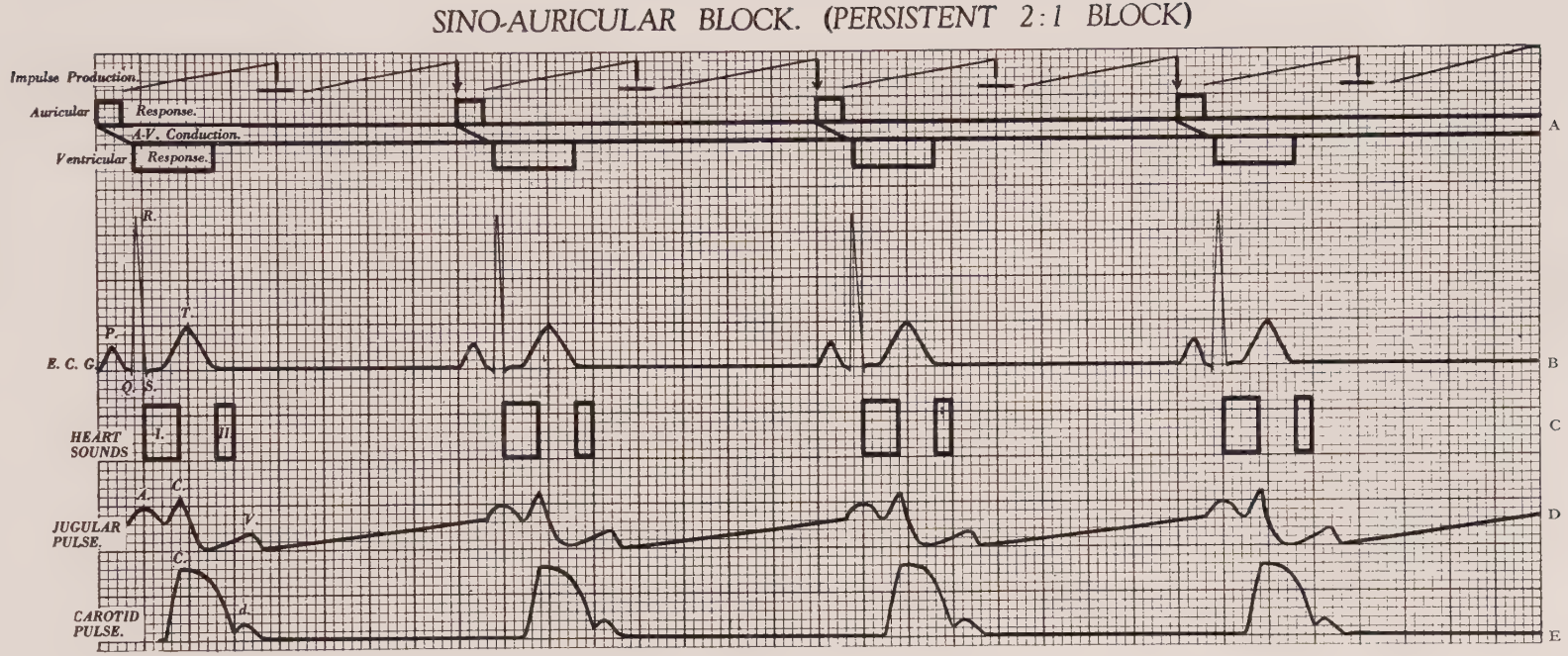
SINO-AURICULAR BLOCK. (PERSISTENT 2:1 BLOCK)
Impulse Production.
Auricular
Response.
A-V. Conduction.
Ventricular
Response.
A
R.
T.
P.
E. C. G.
Q. S.
B
HEART SOUNDS
I.
II.
C
A.
C.
V.
JUGULAR PULSE.
D
C.
d.
CAROTID PULSE.
E

FIG. 26.

"sino-auricular block," as a possible cause of the bradycardia, may be excluded.

Nodal Rhythm. "Sino-auricular block" may at times be of such a marked degree that the lengths of the slow phases may approximately measure three normal cycles. In such hearts, the slow auricular rate may permit an occasional escape of a stimulus from a lower center of impulse production, usually from the auriculo-ventricular node. At times, there may be numerous "nodal ecapes," as a result of which the slow heart beat is rendered also irregular. When they occur in large numbers, the ventricles may beat persistently, for the time being, in response to stimuli from the auriculo-ventricular node. Two pacemakers may thus be acting independently for varying periods. This form of disturbance is extremely rare and cannot be diagnosed by simple clinical means. Even the electrocardiographic curve is difficult to decipher at times.

The condition may advance to a stage in which, because of a high-grade functional impairment or perhaps because of some pathological lesion involving the sino-auricular node, the pacemaker is at a *complete standstill.* In such a case a secondary center, usually the auriculo-ventricular node, assumes the rôle of pacemaker for the whole heart. This node, being situated at the junction of the upper and lower cardiac chambers, sends its stimuli in two directions, upward to the auricles, and downward to the ventricles, thus activating both these chambers practically at the same time. Graphic records actually show that in such hearts auricles and ventricles beat almost synchronously; the P and R waves in the electrocardiogram and the A and C waves in the polygram are often seen superimposed. Because of the fact that the A-V node sets the pace for the whole heart, this disturbance of cardiac mechanism is referred to as a "nodal rhythm."

Common Clinical Features of the Sinus Arrhythmias

The sinus arrhythmias as a group present some common clinical features. Correlated study of heart sounds and of arterial and venous pulses reveals the fact that during the long diastoles incident to slowing in these disorders, there is no evidence of any dynamic event within the heart. *The entire heart is silent during the slow phases.* Factors which accelerate the heart tend to eliminate these arrhythmias, while influences that slow the heart tend to augment them.

CLINICAL FEATURES OF THE SINUS ARRHYTHMIAS

	Hemodynamics during Slow Phases			Special Features
	Heart Sounds	Jugular Pulse	Arterial Pulse	
Respiratory Sinus Arrhythmia	Absent	Absent	Absent	Groups of rapid heart beats alternating with groups of slow beats. Alternations corresponding to phases of respiration. Common arrhythmia in infancy, puberty, adolescence and during convalescence.
Phasic Sinus Arrhythmia	Absent	Absent	Absent	Periods of gradual slowing of the whole heart. Slowing not associated with respiration. Encountered during digitalis medication, following exercise, and in vagotonia.
Sino-auricular Block	Absent	Absent	Absent	Occasional "dropped" beats intercepting an apparently normal rhythm, or a bradycardia which doubles its rate on exercise. Seen in rheumatic fever, during digitalis medication, and after administration of quinidine.

These arrhythmias tend to subside, at times even to disappear, on acceleration of the heart rate from any cause: exercise, fever, amyl nitrite, atropine, etc. They tend to be augmented by vagotropic drugs or toxins; digitalis, pilocarpine, morphine, etc.

Chapter VII

Auriculo-ventricular Heart Block

A BRIEF review of the order and the rate of impulse transmission in the normal heart is particularly helpful as an introduction to the study of auriculo-ventricular heart block, because as the name "heart block" implies, in this group of disorders, we are concerned essentially with a disturbance in the transmission of the cardiac impulse. The qualifying phrase "auriculo-ventricular" merely serves to restrict our study to a group in which the primary impairment of function is localized in that portion of the specific system which joins auricles and ventricles; namely, the A-V junction (A-V node and bundle).

Reviewing the normal, we are reminded that the cardiac pacemaker sets a rhythmic tempo for the auricles and that the ventricles in turn follow the auricles in an orderly manner. We are particularly impressed with the fact that normally the auriculo-ventricular conduction time is of a remarkably constant duration in each successive cardiac cycle. This constancy implies, of course, that the path of the impulse between auricles and ventricles, across the A-V junction, is functionally intact.

Studying the rate of impulse transmission in the heart, we further observed that even normally there is a relative delay in transmission at the auriculo-ventricular node. The time that normally elapses between the beginning of auricular systole and the beginning of ventricular systole has been found to vary between 0.13 and 0.20 seconds, as expressed by the P-R interval in the electrocardiogram and the A-C interval in the phlebogram.

Mechanism of Auriculo-Ventricular Heart Block

Conditions may arise that will accentuate the normal delay of the cardiac impulse at the A-V junction; that is, because of some functional or anatomical impairment within the substance of the A-V node or in tissues adjacent to it, impulse transmission may become unduly retarded in this portion of the specific system. Actually, such conditions do arise, not at all infrequently, leading to what is known as auriculo-ventricular heart block. The impairment, to be sure, may be very mild and the slight conduction defect caused by it may pass entirely undetected, except in cases where graphic measures are instituted.

Graphic records in mild disturbances may merely show that the interval designating A-V conduction time is slightly prolonged and that the *degree of prolongation is persistently the same for all cycles.* They may show, for instance, that the conduction time consistently measures 0.24, 0.28 or more seconds in each cardiac cycle, instead of 0.20 seconds which is generally regarded as the upper limit of normal.

On the other hand, conduction may become impaired in a gradual manner so that successive auricular stimuli find it more and more difficult to bridge the junctional tissues. In such a case the *impulse is progressively retarded from cycle to cycle.* This progressive impairment eventually leads to an occasional stage of absolute retardation—an occasional complete interception of the impulse. The auricular stimulus that arrives at the A-V junction at this time entirely fails to influence the ventricles. The path of the impulse is temporarily blocked at the A-V junction and a ventricular beat is therefore "dropped" out of the cardiac cycle. Because of the block and the coinci-

dentally dropped ventricular beat, there occurs a long pause, the duration of which corresponds approximately to two cardiac cycles. During this pause, because of a sufficiently long period of rest, the junctional tissues generally tend to recover their conductivity, so that the conduction time in the cycle immediately following the pause is quite normal. In the subsequent beats, however, progressive retardation is again in evidence, leading after a few cycles to another dropped ventricular beat. The frequent recurrence of these phenomena, that is the dropping of a ventricular beat every now and then, leads to an irregularity of the heart beat.

Graphic records show that the individual complexes of the electrocardiograph and pulse tracings are generally normal in outline. It is the intervals designating auriculo-ventricular conduction time (P-R interval in the electrocardiogram and A-C interval in the venous pulse) that are altered; it is these that show a gradual prolongation from cycle to cycle eventually leading to blocked auricular complexes.

These types of disorder, namely, the persistent low-grade delay in A-V conduction on the one hand, and the progressive retardation leading to dropped beats on the other, characterize the *milder forms of partial auriculo-ventricular heart block* (Fig. 27).

The path of the cardiac impulse at the A-V junction may be further impaired so that, instead of an occasional dropped ventricular beat, the junctional tissues may consistently intercept every third, every second or every second and third supraventricular impulse. Accordingly, depending upon the relative number of auricular contractions to the ventricular contractions, the disturbance may be designated as a 3:2 block, 2:1 block or 3:1 block.

The 2:1 ratio is the most common of the three forms mentioned. It is characterized by a slow rate (40 to 50) and a perfect regularity of rhythm (Fig. 28).

The last mentioned types of A-V conduction disturbances constitute the more *advanced forms of partial auriculo-ventricular heart block.*

As a result of high-grade functional impairment or perhaps an actual anatomical lesion, the A-V junction may completely fail to transmit any of the auricular impulses: all such impulses are intercepted before they reach the ventricles. Communication between auricles and ventricles is, therefore, completely blocked so that there is a "dissociation" of rate and rhythm between upper and lower cardiac chambers. The auricles beat in response to normal, rhythmic stimuli emanating from the site of the cardiac pacemaker; while the ventricles, on the other hand, are activated by impulses elaborated at an ectopic focus, a secondary center of impulse production. It is some portion of the ventricular division of the bundle of His that sets the pace for the ventricles; and the intrinsic ventricular rhythm in a complete dissociation is therefore spoken of as an "idio-ventricular rhythm." The resulting ventricular rate is naturally very slow, 35 or less per minute, but generally regular.[1] Because of the complete dissociation of auricles and ventricles and the consequent idio-ventricular rhythm, this altered cardiac mechanism is designated as a *complete auriculo-ventricular heart block* (Fig. 29).

Such disturbance may at times be functional and therefore merely transitory. More often, however, the underlying defect being organic, such dissociation is permanent, indicating that

[1]The inherent rhythmicity of a secondary center of impulse production is slower than that of the normal cardiac pacemaker.

FIG. 27.—Diagrammatic representation of the mechanism and extracardiac manifestations of Auriculo-Ventricular Heart Block (progressive delay in conduction with an occasional dropped beat).

A, *Mechanism:* Rhythmic impulse production: rhythmic auricular responses. Gradual delay in A-V conduction from beat to beat, leading to an occasional interception of the impulse (dropped beats) as seen in cycle 5.

B, *E. C. G.:* Gradual prolongation of P-R interval from beat to beat leading to occasional block. (Time: $(P\text{-}R)_1 = 0.16$ sec. $(P\text{-}R)_2 = 0.20$ sec. $(P\text{-}R)_3 = 0.24$ sec. $(P.R)_4 = 0.32$ sec. P_5 stands alone ; it is blocked.)

C, *Heart Sounds:* Apparently rhythmic sets of heart sounds with but a slight delay from beat to beat, leading to an occasional pause.

D, *Jugular Pulse.* Gradual delay in the A-C. intervals leading to an *isolated* A *wave* (A_5), at a time when neither heart sound is heard nor arterial pulse felt.

E, *Carotid Pulse:* Apparently normal, rhythmic pulse beats interrupted by an occasional pause (cycle 5). The pulse following the pause is large.

PARTIAL AURICULO-VENTRICULAR HEART BLOCK.

(Gradual delay in conduction with an occasional dropped beat)

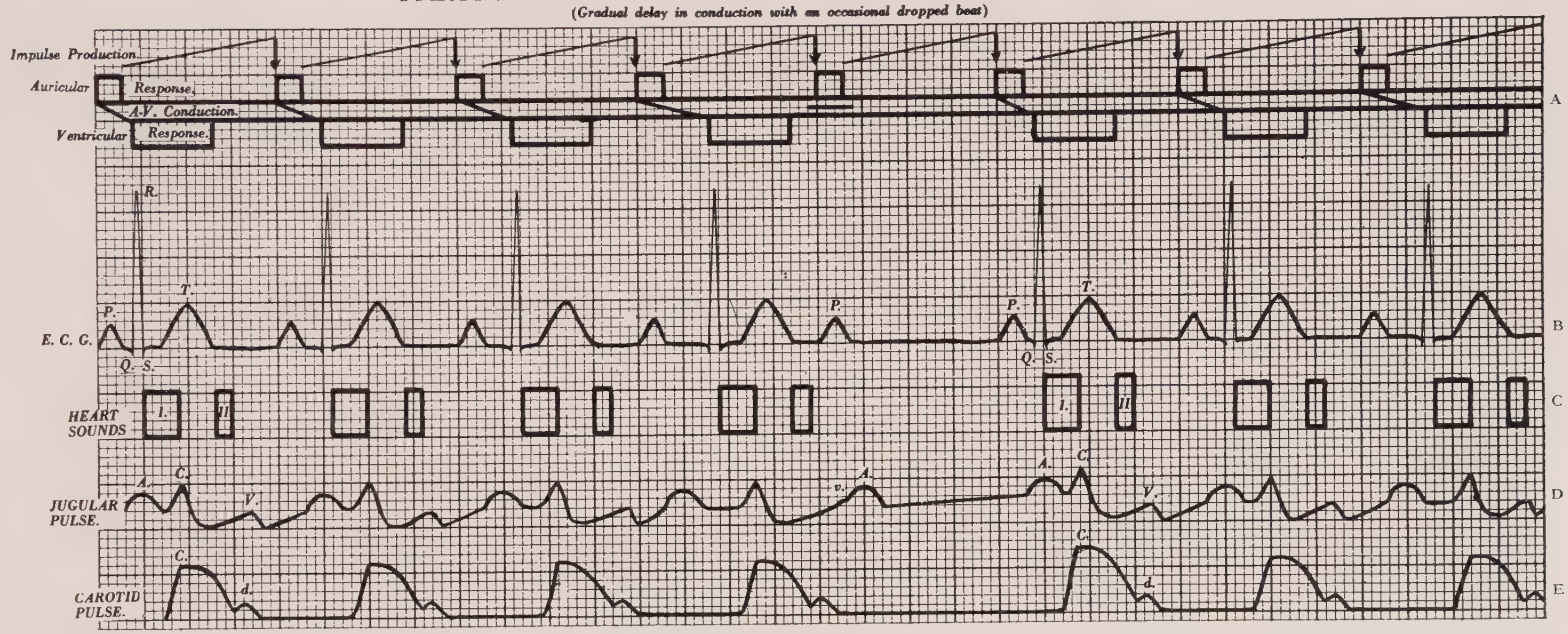

FIG. 27.

FIG. 28.—Diagrammatic representation of the mechanism and extracardiac manifestations of Auriculo-Ventricular Heart Block (2:1).

A, *Mechanism:* Rhythmic impulse production: rhythmic auricular responses. Alternate auricular stimuli are blocked at the A-V junction causing a rhythmic bradycardia of the ventricles.

B, *E. C. G.:* Slight prolongation of P-R interval: in alternate cycles the P waves are blocked as in P_2, P_4, P_6, and P_8.

C, *Heart Sounds:* Heart sounds appear slowly and regularly.

D, *Jugular Pulse:* Solitary A *waves* are seen during the long diastoles when neither heart sounds are heard nor arterial pulse felt.

E, *Carotid Pulse:* Slow and regularly spaced pulse beats.

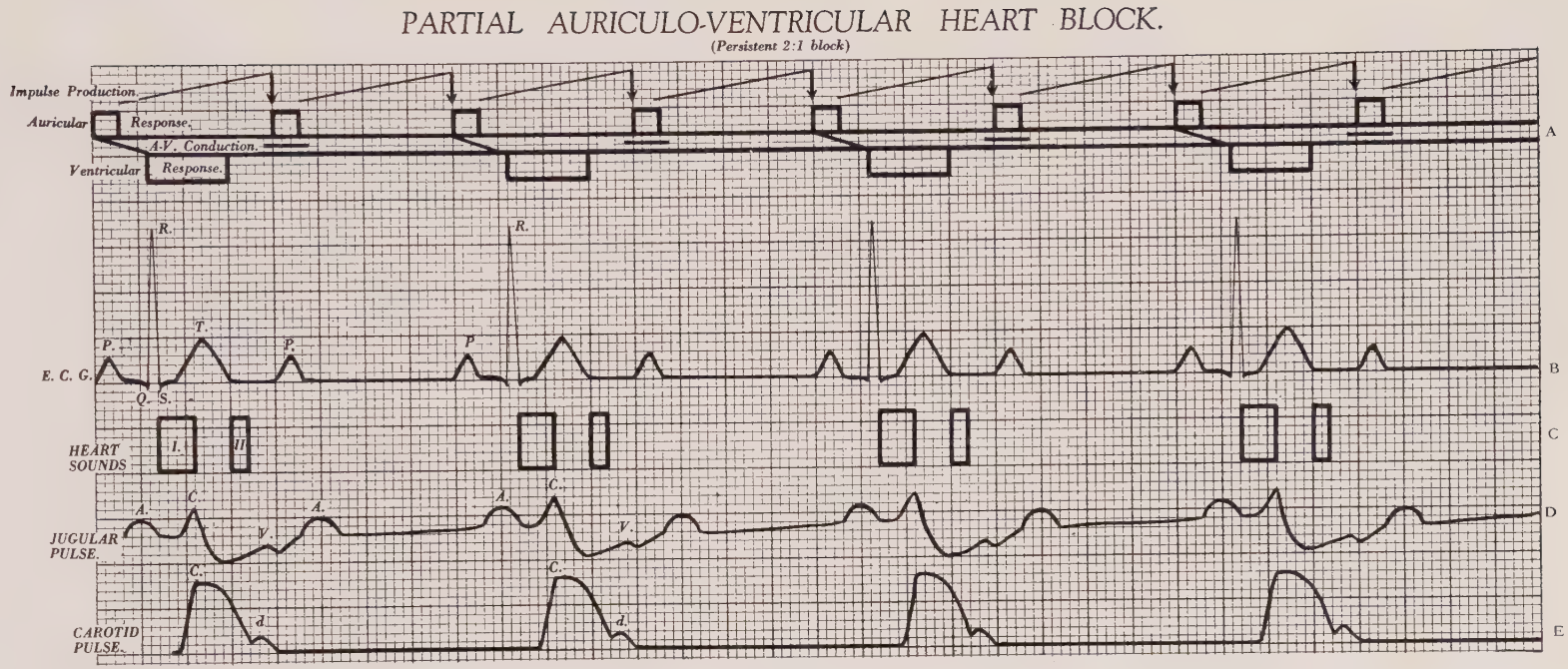
PARTIAL AURICULO-VENTRICULAR HEART BLOCK.
(Persistent 2:1 block)
Impulse Production.
Auricular
Response.
A-V. Conduction.
Ventricular
Response.
A
R.
R.
T.
P.
P.
P
P
E. C. G.
Q.
S.
B
HEART SOUNDS
I.
II
C
A.
C.
A.
A.
C.
V.
V.
JUGULAR PULSE.
D
C.
C.
d.
d.
CAROTID PULSE.
E

FIG. 28.

FIG. 29.—Diagrammatic representation of the mechanism and extracardiac manifestations of Complete Auriculo-Ventricular Heart Block.

A, *Mechanism:* Rhythmic impulse production: rhythmic auricular responses. There is complete interception of all impulses at the A-V junction. The ventricles beat slowly and regularly to an "idio-ventricular rhythm." There is no numerical relation between auricular and ventricular responses.

B, *E, C, G.:* P-P interval = 4/5 sec. R-R interval = 9/5 sec. The P waves bear no relation to the R waves. The two at times accidentally coincide. (Cycles 3 and 4.)

C, *Heart Sounds:* Very slow, regular heart sounds, the characteristics of which are at times slightly altered by faint auricular sounds, now preceding, then following them (not designated in the graph).

D, *Jugular Pulse:* Distint A waves are seen during the long diastoles when neither heart sounds are heard nor arterial pulse felt. Because of the constantly varying relation of the auricular to the ventricular contractions, the venous pulse beats change their form from beat to beat. When the upper and lower chamber contractions coincide, the corresponding venous pulse is unusually large (C and A superimposed in Cycle 4).

E, *Carotid Pulse:* Slow, regular arterial pulse.

COMPLETE AURICULO-VENTRICULAR HEART BLOCK.

(Dissociation of auricles and ventricles)

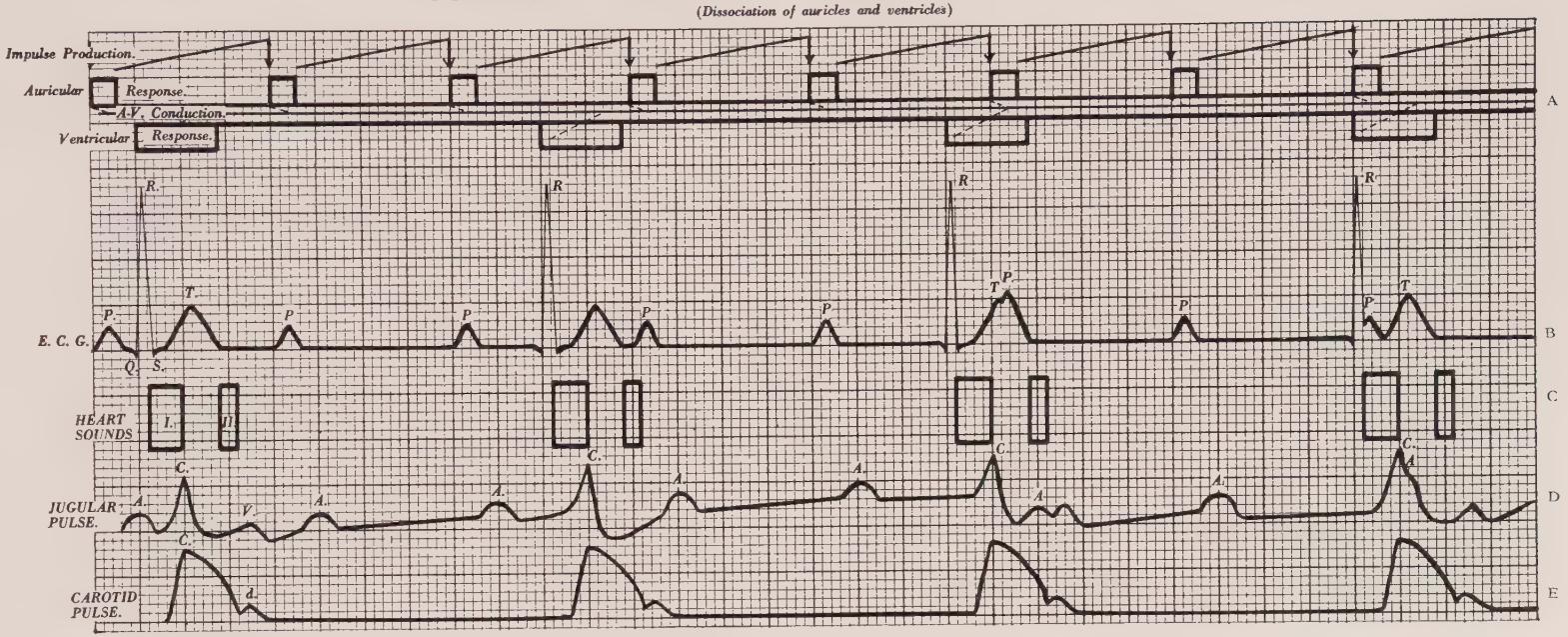

FIG. 29.

there is a permanent anatomical severance between auricles and ventricles, probably resulting from a lesion at or near the bundle of His. Not infrequently, such lesion is but a part of a more extensive myocardial disease.

Because of a widespread disease of the myocardium, the secondary center (idio-ventricular center) setting pace for the ventricles may occasionally show an inconstancy in its performance. Its automaticity for impulse production may at times fail for longer or shorter periods, so that the resulting ventricular rate may occasionally become profoundly slow and irregular, 20 or less per minute (Fig. 30). Marked clinical symptoms and signs may accompany such a disturbance.

Conditions That May Induce Auriculo-ventricular Heart Block

1. acute infections

Acute infections are not infrequently accompanied by the milder forms of A-V heart block. They are especially encountered in the course of acute rheumatic carditis. The term "carditis" is used to imply that it is but on rare occasions that a single layer of the heart is alone involved. It is inconceivable that a pericarditis or endocarditis may go on for any length of time without involving the adjacent myocardium.

Other acute infections of great severity, such as typhoid, pneumonia and influenza or, as in the case of children, scarlet fever or diphtheria, may induce auriculo-ventricular heart block.

2. digitalis administration

Digitalis administration, if carried to toxicity, is not infrequently associated with A-V heart block. It may be justly

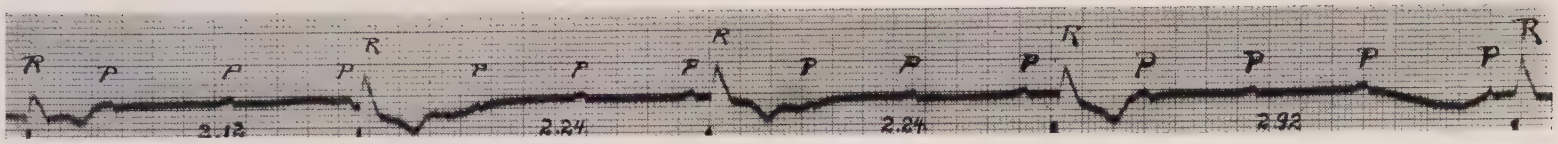

FIG. 30.—Irregular "idio-ventricular rhythm." The ventricular beats are very slow and irregular. Many auricular P waves are seen between the widely spaced ventricular Q,R,S, T complexes. The "idio-ventricular" center of impulse production is depressed and unstable as shown by the progressive lengthening of the interventricular (R-R) periods. (From a case of Stokes-Adams syndrome.)

questioned whether the frequency of heart block in rheumatic carditis is not at least occasionally superinduced by an injudicious administration of digitalis.

The types of heart block usually met with in the before mentioned conditions are the several grades of partial auriculo-ventricular heart block.

3. CHRONIC DEGENERATIVE PROCESSES

Persistent heart block is encountered in a considerable number of elderly people. It is commonly observed that such disturbance is but a part of a more general, sometimes widespread, degenerative process in the heart muscle. Some cases can, no doubt, be traced to repeated attacks of rheumatism or may perhaps be the direct result of syphilis. The most frequent causes of complete auriculo-ventricular heart block in the aged, however, are chronic changes in the heart muscle or in the coronary arteries, such as chronic inflammations, fibrotic, calcific and atheromatous changes.

4. POSSIBLE CONGENITAL ORIGIN

Occasionally a complete auriculo-ventricular heart block is met with in very young children in whom there is no definite history of an etiological factor. The heart block is probably of congenital origin.

CLINICAL FEATURES OF THE VARIOUS TYPES OF AURICULO-VENTRICULAR HEART BLOCK

The several grades of A-V heart block are accurately deciphered by instrumental means. The polygraph and especially the electrocardiograph depict them unmistakably as disturbances in the sequential propagation of the cardiac impulse.

Since, however, the purpose of this book is essentially a clinical one, its object being to render these anomalies comprehensible by aid of simple clinical signs, particular stress is laid upon those clinical features which may be detected by the special senses.

1. PERSISTENT DELAY IN AURICULO-VENTRICULAR CONDUCTION

This is the mildest form of heart block. It is the result of slight or moderate interference with impulse transmission. It causes no irregularity of the heart beat and can but rarely be recognized by ordinary clinical means. In graphic records (especially the E.C.G.) the A-V conduction time is found definitely prolonged and the degree of the prolongation is generally constant for all cycles (Fig. 31).

Even in these, however, the experienced or enthusiastic clinician may at times glean some information from the auscultatory findings.

Auricular sounds are normally inaudible. This is due, no doubt, to the fact that the faint auricular sounds practically merge with the loud, ventricular first heart sounds. In the early form of heart block, however, there being a delay in auriculo-ventricular conduction, auricular contraction precedes the ventricular contraction by an appreciable interval, so that the auricular sound is definitely separated from the more distinct, loud ventricular sound. In such a case, an attentive listener may detect the faint auricular sounds as fine, muffled presystolic reduplications of the first heart sounds. If A-V conduction is much delayed, the muffled auricular sound may appear rather early in diastole; so early at times as to follow closely upon the second sound of the preceding heart beat and

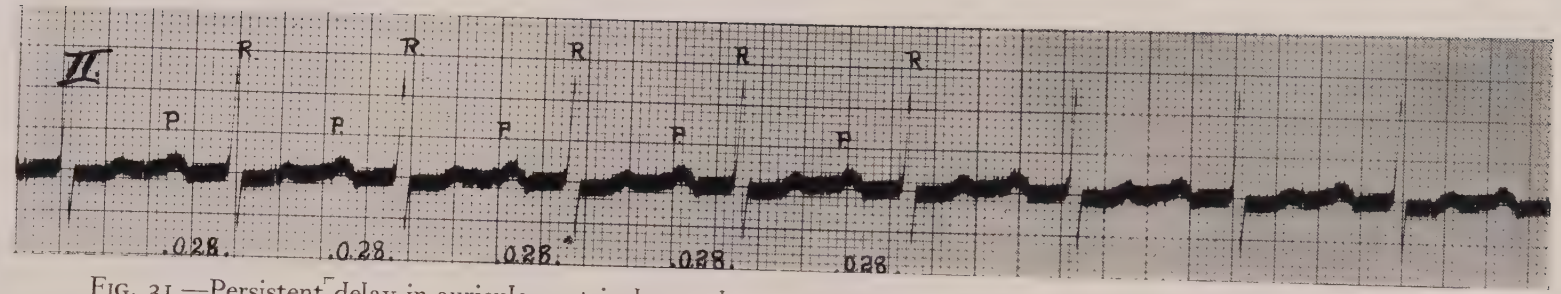

FIG. 31.—Persistent delay in auriculo-ventricular conduction. The rate and rhythm of the heart are normal. The P-R intervals are considerably prolonged, but the prolongation is practically of the same degree in each successive cycle (P-R interval = 0.28 seconds).

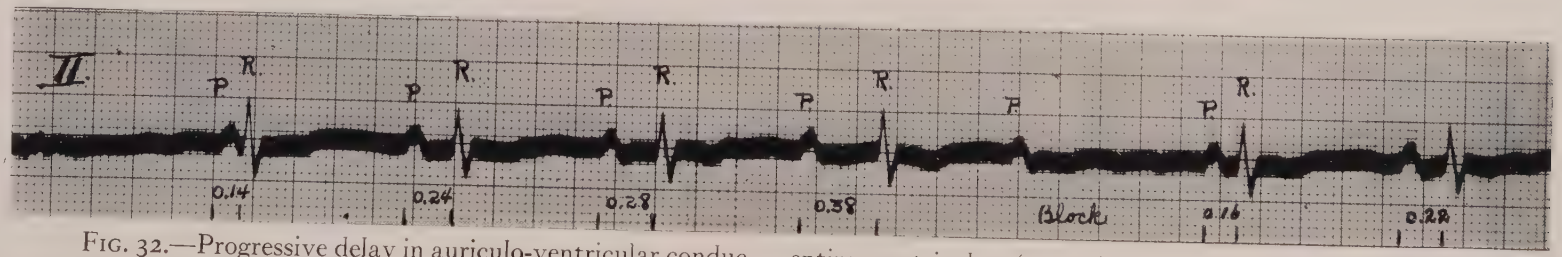

FIG. 32.—Progressive delay in auriculo-ventricular conduction from cycle to cycle, leading to an occasional "dropped beat." $(P\text{-}R)_1 = 0.14$ sec. $(P\text{-}R)_2 = 0.24$ sec. $(P\text{-}R)_3 = 0.28$ sec. $(P\text{-}R)_4 = 0.38$ sec. P_5 is blocked and at this point an entire ventricular (Q,R,S,T) complex is "dropped" out of the cardiac cycle. $(P\text{-}R)_6$ shows the return to a temporary normal conduction.

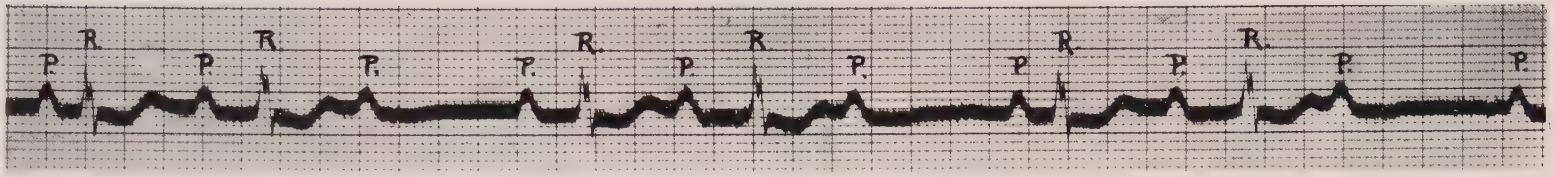

FIG. 33.—3:2 partial auriculo-ventricular heart block. Every third auricular (P) wave is consistently blocked, so that the *ratio* between auricular and ventricular contractions is 3 to 2.

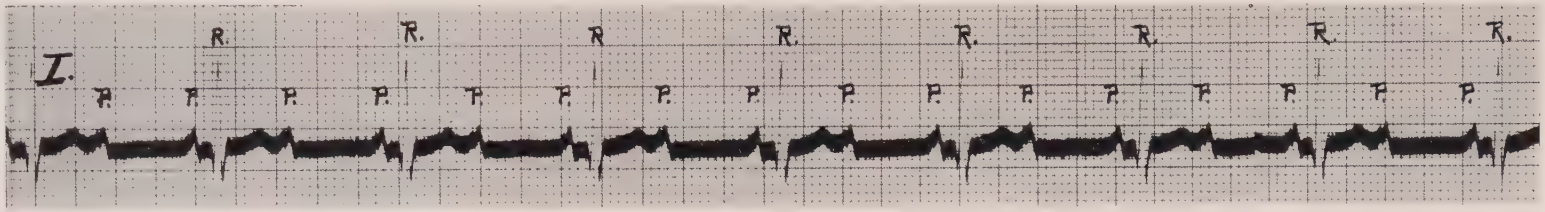

FIG. 34A.—2:1 partial auriculo-ventricular heart block.

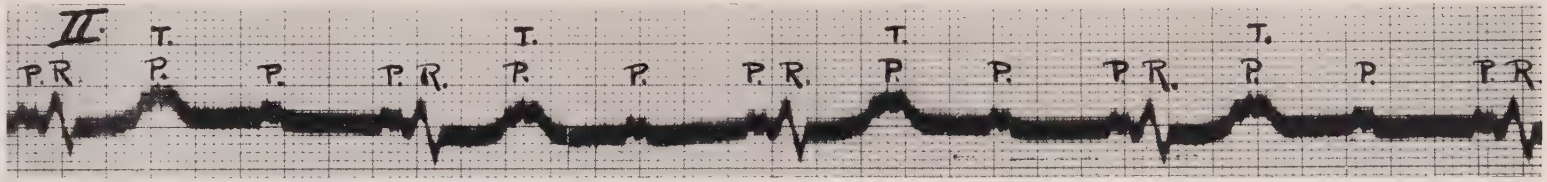

FIG. 34B.—3:1 partial auriculo-ventricular heart block.

thus cause a faint reduplication of the second heart sound instead of the first ("gallop rhythm").

If mitral stenosis is present, these findings become more evident. Auricular contractions in mitral stenosis are accompanied by an apical thrill and rumble which ordinarily gradually merge into the first heart sound. If the auriculo-ventricular conduction time is moderately prolonged, the rumble may be heard to precede the first heart sound by an appreciable interval; if markedly prolonged, the rumble may fall so far behind the first heart sound as actually to follow closely upon the second sound of the preceding systole. The relative appearance of the rough, presystolic, stenotic murmur in the cardiac cycle at times helps to disclose the nature of the disorder with considerable accuracy as a mild form of auriculo-ventricular heart block.

While these clinical signs are by no means easily detected, they may, nevertheless, often serve to suggest the presence of the anomalous cardiac condition, so that timely graphic measures may be instituted.

2. PROGRESSIVE DELAY IN AURICULO-VENTRICULAR CONDUCTION LEADING TO OCCASIONAL DROPPED BEATS (FIG. 32)

This condition differs from the former in that here the A-V conduction is generally gradually prolonged from cycle to cycle until it eventually culminates in a "dropped" ventricular beat. On auscultation, one may note the faint but varying shades of reduplication of the first and second heart sounds from cycle to cycle, as a result of the constantly varying relation of the auricular to the subsequent ventricular contractions. In addition, however, because of an occasional "dropped beat," the apparently regular cardiac rhythm is interrupted by a

pause on the part of the heart sounds and the arterial pulse, suggesting an occasional temporary silence of the whole heart. Actually, however, only the ventricles are silent. Careful visualization of the jugular vein during such pause reveals a distinct pulsation, indicating that the auricles are not partaking in the pause.

Dropped beats, when frequent, at times recur regularly. Figure 33 is an example of such a grouping. It shows a dropped beat in every third cycle, giving rise to a 3:2 auriculo-ventricular ratio (3:2 A-V partial heart block). Clinically such a heart presents a coupling of the heart sounds and of the arterial pulse. During the pauses, isolated pulsations of the jugular vein may be seen.

Exercise, because of its tendency to accelerate the heart rate, eliminates the pauses and the heart becomes regular. On rest *subsequent to exercise* the pauses tend to reappear. Atropine administration may have an effect similar to exercise.

3. THE 2 TO 1 OR 3 TO 1 PARTIAL AURICULO-VENTRICULAR HEART BLOCK (FIG. 34 A AND B)

These conditions represent a further advanced impairment over the former disturbance. Here every second, or every second and third impulse is consistently blocked at the A-V junction. The resulting ventricular response is, therefore, *regular* and comparatively *slow*. If the block is 2:1, the heart rate is generally between 40 and 60. If the block is 3:1 (rare), the rate is between 30 and 40. Because of the regular block and the consequent, slow, regular ventricular rate, these disorders manifest themselves clinically as bradycardias.

The clinical feature that characterizes the slow phases of the aforementioned arrhythmia (progressive delay in A-V con-

duction leading to occasional dropped beats) is present in all the cycles of these bradycardias, namely isolated jugular pulsations may be seen during the long pauses of apparent cardiac standstill when there are neither heart sounds heard nor arterial pulse felt. This clinical feature distinguishes these bradycardias from the so-called simple bradycardias.

The clinical recognition of 2:1 heart block is much facilitated if mitral stenosis co-exists, because, there being two auricular beats to each ventricular beat, two presystolic rumbles may be heard in each of the long diastoles; one far removed from, and one immediately preceding, the first heart sound.

Exercise tends to remove the bradycardia of 2:1 heart block and transform it either to a regular rhythm, the rate of which is double the rate of the bradycardia, or into an irregularity with dropped beats. Atropine also tends to eliminate the bradycardia.

4. COMPLETE AURICULO-VENTRICULAR HEART BLOCK (FIG. 29)

This disturbance is characterized by marked slowing of the ventricles, usually below 35 per minute. The rhythm is generally regular. The first and second heart sounds appear consistently, but careful auscultation reveals the interesting fact that the heart sounds tend to be somewhat altered in character from one beat to another. A faint, muffled sound may now precede and then follow either the first or second heart sound. This is due to the independent auricular contractions which by mere chance may fall into any part of the long ventricular diastoles. When one of these auricular contractions happens to fall at the time of ventricular systole, when the regular first heart sound occurs, it is apt to modify the intensity or character of the first or second heart sound considerably (Fig. 35).

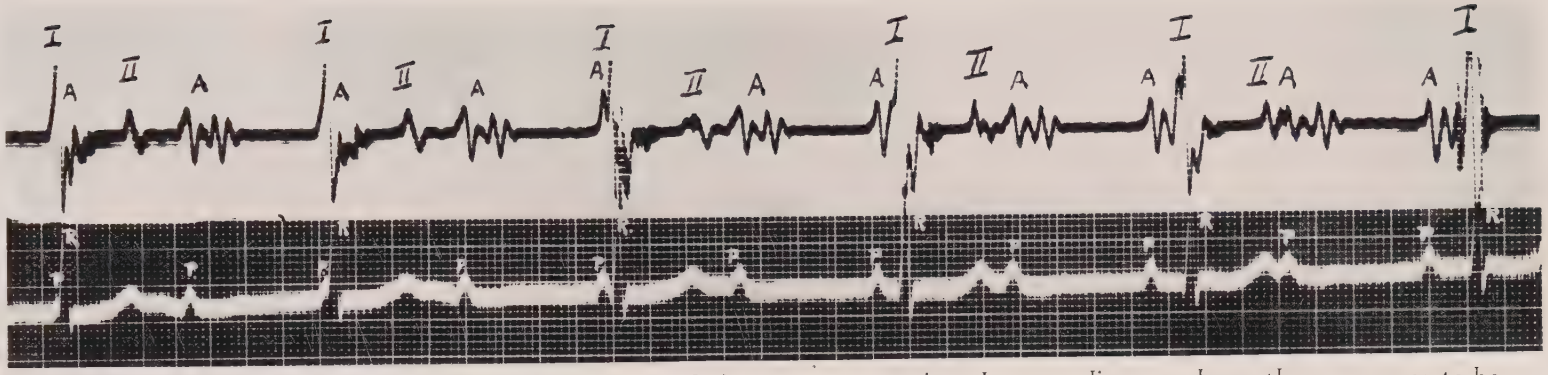

Fig. 35.—Synchronous tracing of heart sounds and electrocardiogram in a case of complete A-V heart block, showing how the auricular sounds (A) modify the normal first and second heart sounds successively from beat to beat. The accompanying electrocardiogram shows these A waves to be synchronous with auricular activity and thus discloses their identity as being auricular sounds.

The jugular pulse in complete auriculo-ventricular heart block may also present a unique feature. Since the number of auricular contractions bears no direct relation to the number of ventricular contractions and since they may therefore at times appear together and at other times far apart, the jugular vein may manifest in this condition a very varying type of pulsation (Fig. 36). In contradistinction to the normal venous pulse in which the A, C, V waves follow each other and in which visualization reveals recurrent rhythmic series of fine wavelets, the jugular pulse in complete A-V block is seen to vary considerably in size and shape, from beat to beat. At times there is but a single large wave seen; at other times a group appears. At any rate, the type of the venous pulse beat in a given cycle generally bears no resemblance to the one preceding or to the one following it. At times it is seen to become progressively larger in successive cycles.

In auriculo-ventricular heart block both the height of the jugular pulse and the character and intensity of the heart sounds often vary from beat to beat. Both phenomena have the same underlying cause, namely, the persistent dissociation of the upper and lower chamber contractions.

Since the auricles beat two or three times as fast as the ventricles, the contractions of these independent chambers occasionally coincide by mere chance. During such coincidence their effects are naturally summated and it is their combined effect on the hemodynamics of the circulation that gives rise to temporary exaggerations of clinical signs. Thus an occasional heart sound at times appears louder than the rest and an occasional jugular pulse beat more prominent than the others, even though the rhythm of the heart beat and that of the arterial pulse remain unaltered.

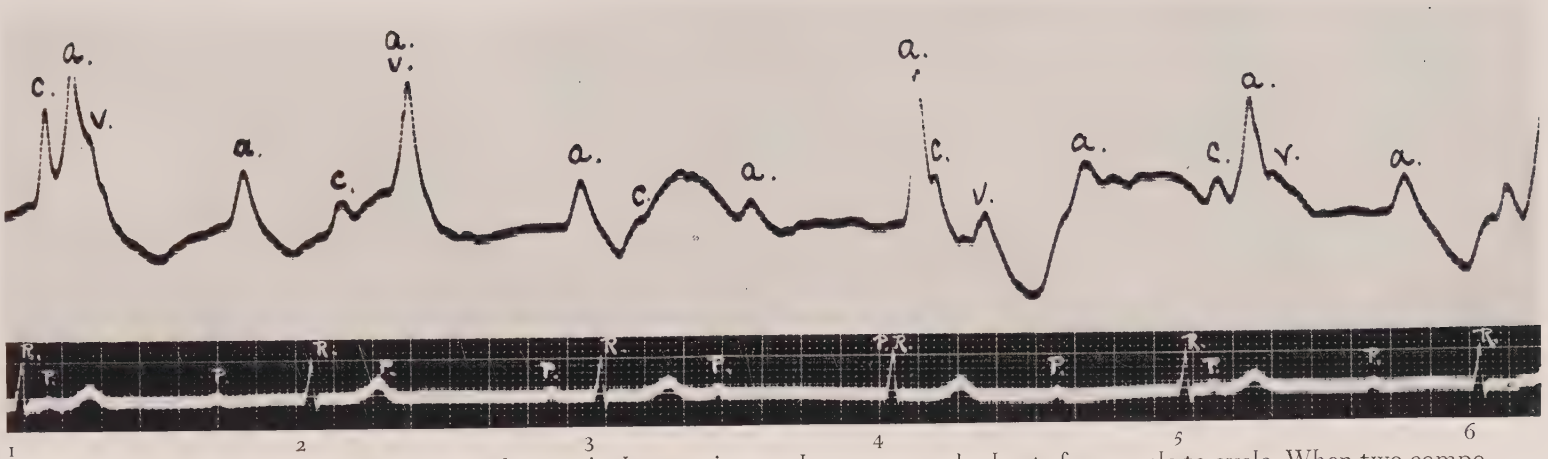

FIG. 36.—The jugular pulse in complete auriculo-ventricular heart block. There is a change in the size and form of the venous pulse beats from cycle to cycle. When two components coincide, the venous pulse is very high (cycle 4).

Whenever we encounter a persistently slow heart action in which the rate is below 35, in which the heart sounds alter their characteristics from beat to beat and in which the jugular veins show a variation in the amplitudes of their pulsations (not due to respiration), the clinical diagnosis of complete auriculo-ventricular heart block is justifiable. Graphic records are conclusive.

In the phlebogram (Fig. 36), the A waves (auricular waves) are rhythmically spaced and their rate is approximately normal. The C waves (ventricular waves) are also regularly spaced but their rate is much slower. There is no definite numerical relationship between the two. As has been mentioned the height of the waves are often seen to vary because of superimposition of two phenomena, giving rise at times to waves of unusual amplitude.

In the electrocardiogram, the auricular P waves are regularly spaced and their outlines are normal except when they chance to coincide with a ventricular complex. The ventricular complexes Q, R, S, T are also rhythmic in their appearance but their rate is slow. The configuration of these complexes is generally normal. At times, however, the initial ventricular complex (Q, R, S) assumes a bizarre outline: that is, the main deflection (R) is unusually large and widened and resembles the so-called bundle branch block type.[1] This may be due either to a defective conduction in one bundle branch or perhaps to the fact that the pacemaker is situated below the bifurcation of the main bundle of His. At times, the ventricular

[1] By bundle branch block is meant an experimental, physiological, or pathological interruption of the impulse path along one of the main branches of the bundle of His. In the electrocardiogram, this condition is characterized by a marked distortion of the initial ventricular deflection. The Q, R, S complex is very tall, notched and much widened. The direction of the terminal deflection T is always opposite to the direction of the initial Q, R, S complex.

complexes may appear unusually stunted, widened and splintered.[1] This may indicate an associated generalized intraventricular conduction defect, often due to an associated extensive atherosclerotic myocardial damage.

The various forms of clinical heart block, because of a predominance of the functional (temporary) type of impediment to conduction across the A-V junction, at times show transitions from one grade of disorder to another. The partial form may progress to the complete and the complete form may revert to the partial.

Complete heart block, while generally regular, may at times become exceedingly slow and irregular (Fig. 31). During such very slow phases, additional clinical signs, due to alterations in the general circulation, may appear. These may be fainting, loss of consciousness, twitching or actual convulsions with stertorous breathing (Stokes-Adams syndrome). Depending on the degree of slowing, either pallor or cyanosis may be present.

[1] This type of ventricular complex in the electrocardiogram has been termed the "arborization block" type. Oppenheimer, B. S., and Rothschild, M. A. Abnormalities in the QRS group of the electrocardiogram associated with myocardial involvement. Preliminary report. *Proc. Soc. Exper. Biol. & Med.*, 1916, xiv, 57–59.

CLINICAL FEATURES OF THE AURICULO-VENTRICULAR HEART BLOCKS

	Hemodynamics during Slow Phases			Special Features
	Heart Sounds	Jugular Pulse	Arterial Pulse	
Persistent delay in A-V conduction	No slow phases.			Faint reduplication of first or, at times, of second heart sounds.
Progressive delay in A-V conduction leading to "dropped beats"	Absent	Present	Absent	Occasional "dropped beat" in an apparently rhythmic heart. Seen in rheumatic fever or in the course of digitalis medication.
2:1 and 3:1 partial heart block	Absent	Present	Absent	Moderately slow and regular ventricular rhythm—a bradycardia of 35 to 60 beats per minute. Seen in rheumatic fever, in diphtheria or in the course of digitalis medication.
Complete heart block	Absent	Present	Absent	Slow, regular heart rate generally below 35 per minute. At times very slow and irregular, associated with syncope. May occur in the course of digitalis medication; in the later stages of untreated diphtheria, and in atherosclerotic myocardial changes.

These arrhythmias tend to subside, at times even to disappear, on acceleration of the heart rate from any cause: exercise, fever, amyl nitrite, atropine, etc. They tend to be augmented by vagotropic drugs or toxins: digitalis, pilocarpine, morphine, etc.

Chapter VIII

Extrasystolic Arrhythmias

(premature beats)

THE rhythmic series of events that characterizes the normal heart beat depends upon a rhythmic series of impulses discharged at the site of the cardiac pacemaker. While the actual time required for the elaboration of the normal cardiac impulse is unknown, it, nevertheless, seems remarkably constant from cycle to cycle. In fact, the tempo of the whole heart depends upon the time required for its production. If impulse production is rapid, the heart rate is rapid. If, on the other hand, impulse production is slow, the heart rate is slow. In either case, the pacemaker normally dominates the situation absolutely. It sets the pace and determines the rate and rhythm of the heart beat.

Occasions may arise, however, in which the normal rhythmic series of events is interrupted by a premature heart beat, induced by an impulse elaborated prematurely at some focus other than the normal cardiac pacemaker. Such "ectopic" stimulus is built up rapidly and is generally discharged before the completion of the sinus impulse belonging to the normal rhythmic series. Being built up prematurely, such an impulse initiates a cardiac response which is also untimely and which, therefore, anticipates the heart beat of the rhythmic series about to be induced by the normal pacemaker and thus disturbs the uniform sequence of events that characterizes the normal heart beat. The premature cardiac contractions thus induced are termed *extrasystoles* or premature beats.

Premature impulses may be elaborated in the ventricles, in the auricles or in the junctional tissues. Depending upon the site of their origin they are classified as ventricular, auricular or nodal extrasystoles. The last-mentioned is an extremely rare type and, for purely clinical purposes, may be disregarded. Its recognition is rendered possible only by accurate graphic records, such as those yielded by the electrocardiograph.

Mechanism of Ventricular Extrasystoles (Fig. 37)

In the normal rhythmic heart, the tempo of ventricular contractions is determined by the tempo of the auricular contractions which in turn depend upon the rate of impulse production at the cardiac pacemaker. The ventricles follow the auricles with a remarkable constancy. The time interval that separates their respective contractions is constant to the minutest fraction of a second. Even when the auricular rate is slow or irregular, providing A-V conduction is normal, the ventricles usually wait for the impulses coming from the upper chambers.

Occasionally, however, as a result of undue irritability in some focus within their own substances, the ventricles may give rise to a premature stimulus in response to which they contract prematurely, or "out of step." Having contracted and having no other premature impulse to activate them, the ventricles usually *wait* for the next normal auricular impulse.[1] The premature ventricular contraction is naturally preceded by only a brief diastole. The subsequent "waiting for the auricles," on the other hand, is characterized by an unusually long diastole,

[1]The ventricles, having contracted prematurely, are usually refractory to the normal rhythmic auricular impulse reaching them at this time. By the time they have recovered the auricles are in a state of diastole, and the ventricles, therefore, must wait for the next normal sinus impulse coming to them by way of the next auricular contraction.

which generally *fully compensates* for the brevity of the preceding one. Consequently, this long pause, following a ventricular premature beat, is referred to as the "compensatory pause" (Fig. 37, cycle 3).

At the time when a premature ventricular contraction takes place, the rhythm of the auricular contraction is generally undisturbed, because the auricles beat in response to the rhythmic sinus impulses. Auricular and ventricular beats may, however, coincide; in fact they usually do. The cause of such synchrony of contraction of upper and lower chambers at the time of the premature ventricular beat becomes evident if we remember that normally the auricular beat precedes the ventricular by 0.12 to 0.20 second only, and that, therefore, if a ventricular beat appears ahead of its time, it is apt to concur with the auricular systole which, normally, it ought to follow.

There is, however, an occasional exception to this concurrence. A ventricular premature beat may occur so early in diastole as actually to precede the auricular contraction by an appreciable time interval. In such a case, the recovery of the ventricular musculature may be completed before the auricular stimulus reaches it, so that, in spite of their previous premature contraction, the ventricles can respond again, this time, however, to one of the rhythmic auricular stimuli. Such extrasystole falls between two normally spaced heart beats: it is "interpolated." In this type of extrasystole, the ventricles do not have to wait, and consequently there is no compensatory pause. Interpolated premature beats are true extrasystoles in that they actually *add* a heart beat (Fig. 37, cycle 7).

Ventricular extrasystoles may occur in groups of two or more (Fig. 38, cycles 3 and 4, 7 and 8). Rarely, a premature stimulus arising in the ventricle, besides inducing a premature

FIG. 37.—Diagrammatic representation of the mechanism and extracardiac manifestations of Ventricular Premature Beats (extrasystoles).

A, *Mechanism:* Rhythmic impulse production: rhythmic auricular responses. An occasional premature impulse arising within the ventricles initiates a premature ventricular response (cycle 3 and cycle 7). In cycle 3, the premature ventricular contraction coincides with one of the rhythmic auricular contractions. A compensatory pause follows; the ventricles are *waiting* for the next orderly supraventricular stimulus. In cycle 7, the premature ventricular beat occurs very early in diastole. It does not coincide with an auricular contraction; in fact, it completes its dynamic phase and recovers in time to respond again to the next rhythmic supraventricular impulse. It does not have to "wait," and therefore there is no compensatory pause. It is "interpolated" between two normal beats.

B, *E. C. G.:* Ventricular rhythm is disturbed by two premature bizarre ventricular complexes (tall, widened, notched and the T waves directed opposite the R waves). In cycle 3, a rhythmic P wave coincides with the terminal phase of the premature ventricular complex: it is followed by a pause which fully compensates for the brevity of the pause preceding the extrasystole. P_4 begins the normal rhythmic series of heartbeats. In cycle 7, another premature ventricular complex appears very early; immediately upon the completion of its terminal phase, a P wave appears followed by a normal Q, R, S complex, and the rhythmic series of events again follow. The premature beat in cycle 7 is interpolated.

C, *Heart Sounds:* The rhythmic series of heart sounds are interrupted by premature first and second heart sounds in cycle 3 and 7. The premature first sounds are sharp and of short duration. In cycle 7, the premature second sound is barely heard. The heart sounds in cycle 4, following the pause, are louder than the rest of the normal sounds.

D, *Jugular Pulse:* The venous pulse corresponding to the premature ventricular beat (cycle 3) is large (combined effect of ventricular and auricular contractions transmitted into veins).

E, *Carotid Pulse:* A premature, faint pulse wave in cycle 3 is followed by a pause. The pulse wave following the pause is large. The premature beat in cycle 7 does not influence the arterial pulse—it is ineffectual.

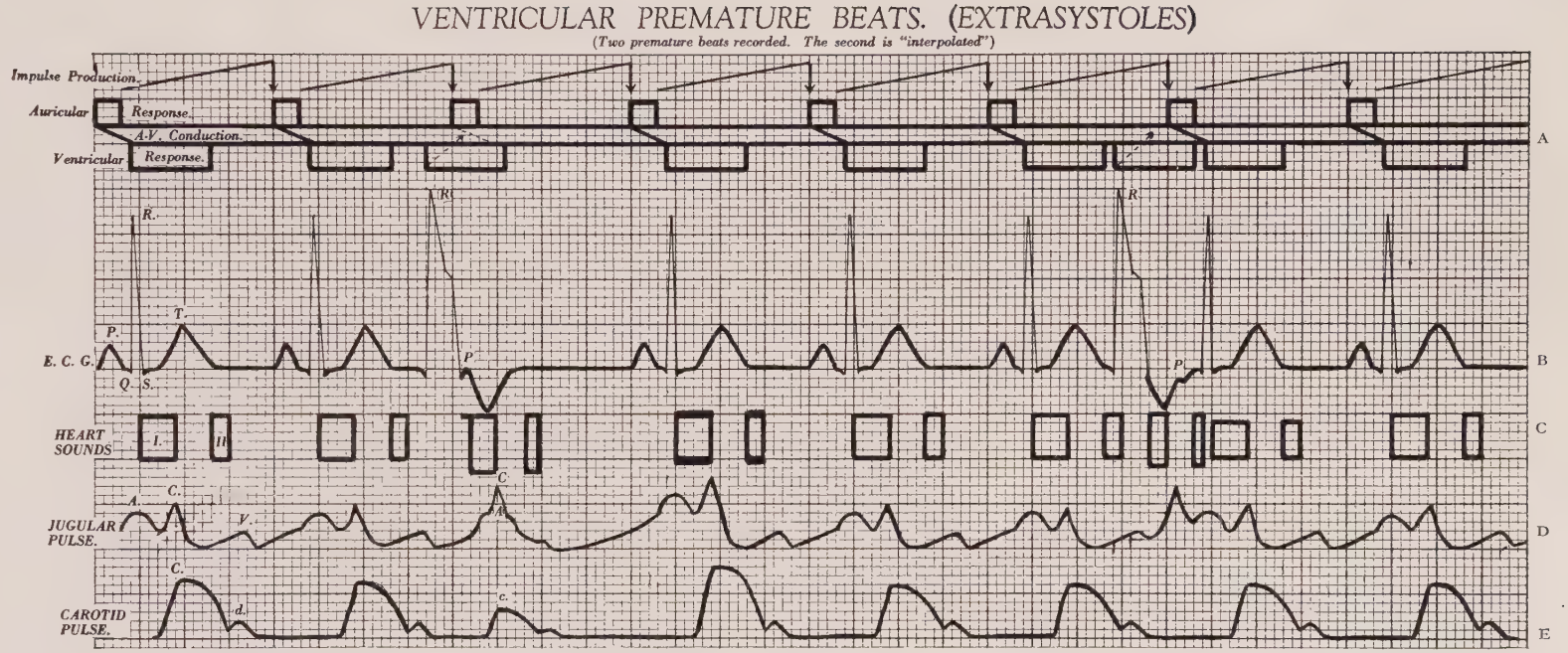
VENTRICULAR PREMATURE BEATS. (EXTRASYSTOLES)
(Two premature beats recorded. The second is "interpolated")
Impulse Production.
Auricular Response.
A-V. Conduction.
Ventricular Response.
A
R.
R'
R
P.
T.
E. C. G.
Q.
S.
P
B
HEART SOUNDS
I.
II
C
A.
C.
V.
JUGULAR PULSE.
D
C.
d.
c.
CAROTID PULSE.
E

FIG. 37.

FIG. 38.—Diagrammatic representation of the mechanism and extracardiac manifestations of Ventricular Premature Beats (grouped extrasystoles).

A, *Mechanism:* Rhythmic impulse production: rhythmic auricular responses up to cycle 7. The ventricular rhythm is disturbed by premature ventricular systoles in cycle, 3, 4, 7 and 8—coupled ventricular extrasystoles. In cycle 3, the ventricular premature beat coincides with an auricular beat: the one in cycle 4 is interpolated. In cycle 7, the extrasystole again coincides with an auricular beat. The one in cycle 8 succeeds in sending a retrograde impulse to the auricles and to induce a premature auricular response, thus temporarily upsetting the rhythm of the upper cardiac chambers.

B, *E. C. G.:* In cycle 3, a premature bizarre Q, R, S, T complex coincides with a P wave. In cycle 4, there is an isolated premature Q, R, S, T of similar type (coupled premature ventricular complexes of which the second is interpolated). P_4 begins the normal rhythmic series of heart beats. In cycle 7, there is a premature ventricular complex, the terminal phase of which coincides with a normal P wave; there is another in cycle 8, within the terminal phase of which there is a premature P wave (coupled extrasystoles of ventricular origin, the second of which induced a premature auricular beat).

C, *Heart Sounds:* The rhythm of the heart sounds is disturbed by premature first and second heart sounds in cycles 3 and 7: and by premature first heart sounds only in cycles 4 and 8. The heart sounds in the last cycle following the long pause are loud.

D, *Jugular Pulse:* Prominent jugular pulse in cycles 3 and 7 when a rhythmic auricular contraction coincides with a premature ventricular contraction.

E, *Carotid Pulse:* Trigeminal rhythm, in which the third pulse beats are feeble and correspond to the first members of the coupled premature beats. The second members of the paired premature beats do not influence the pulse; hence the pauses. The pulse beats following the pauses are large.

VENTRICULAR PREMATURE BEATS. (EXTRASYSTOLES)

(Grouped extrasystoles)

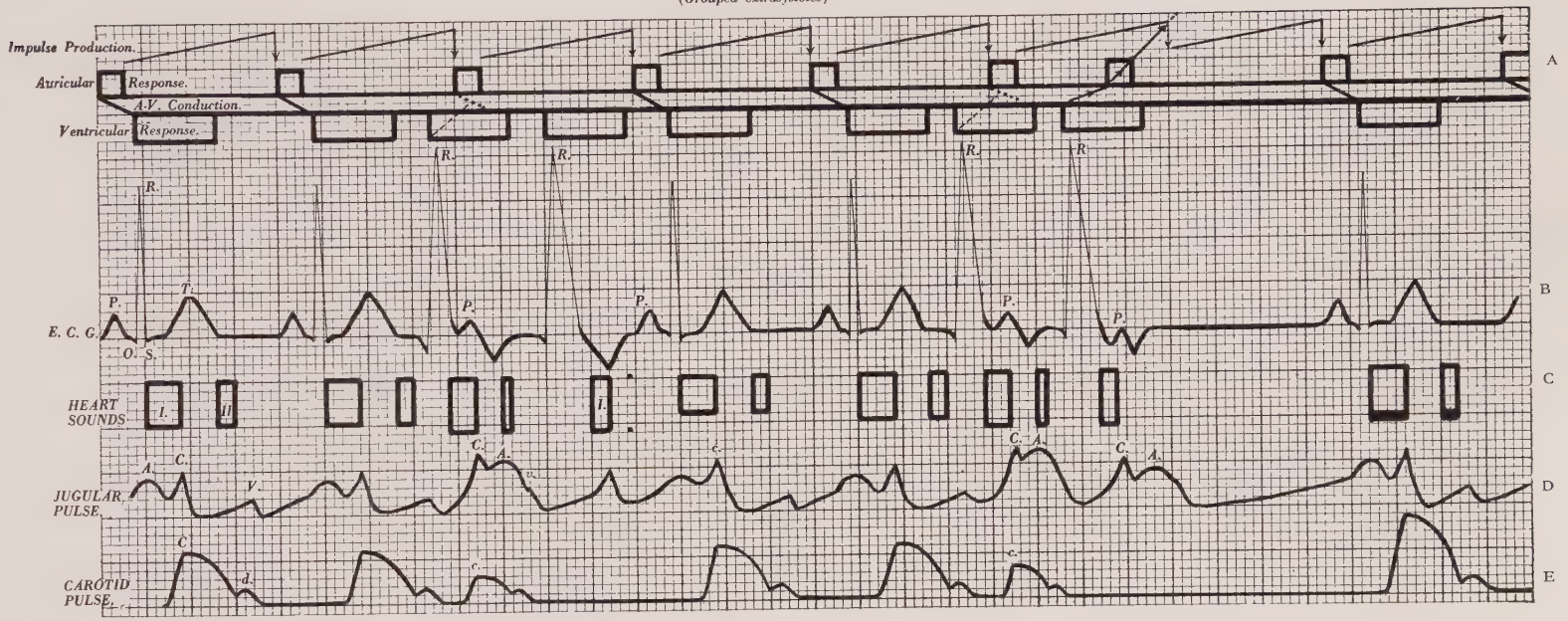

FIG. 38.

ventricular contraction, may also bridge the junctional tissues in a retrograde direction and induce a premature auricular contraction, thus disturbing, for the time being, the rhythm of the auricular beats (Fig. 38, cycle 8).

Mechanism of Auricular Extrasystoles

Ectopic stimuli inducing premature contractions may arise within the substance of the auricles, in situations other than the normal cardiac pacemaker. They may arise high up near the pacemaker, low down near the A-V node, or in foci intermediate between the two. As a group, premature contractions arising any where within the substance of the auricles may be classed as *supraventricular premature beats.*

A premature auricular contraction is generally followed by a ventricular contraction which, of course, is also premature. An *entire* cardiac cycle is thus prematurely induced (Fig. 39, cycle 4, and Fig. 40, cycles 3, 6 and 9).

The *pause* following an auricular premature beat is not fully compensatory, as in the case of ventricular extrasystoles.

The length of the pause following auricular, or, generally speaking, supraventricular premature beats, is at least in part determined by the location of the focus which gives rise to the premature impulse. For instance, if a stimulus arises anywhere in the auricle, outside of the pacemaker, such stimulus spreads throughout both auricles and induces a contraction. In the course of its spread it necessarily also involves that portion of the auricular musculature which corresponds to the region of the sino-auricular node. A short interval elapses before the impulse can spread to the region of the pacemaker. The time required to reach it depends at least in part upon the distance of the ectopic focus from it. Upon reaching the pace-

maker, the premature stimulus annihilates an *immature* sinus impulse, one of the series that is being rhythmically produced at the S-A node. From the moment that such an impulse is destroyed, there begins the formation of a new rhythmic series of impulses by the pacemaker, at the usual rate.

The time it takes for the premature stimulus to reach the pacemaker, together with the time required for the formation of a new impulse of the rhythmic series, approximately equals the time expressed by the compensatory pause following a supraventricular extrasystole.

The time required for the elaboration of the normal sinus impulse is usually constant for a given heart beat. The time utilized for the spread of the ectopic stimulus in order that it may reach the pacemaker is variable however and depends in part at least upon the distance of the ectopic focus from the site of the pacemaker. It becomes evident, therefore, that in auricular extrasystoles, the nearer the ectopic focus to the pacemaker, the shorter the compensatory pause; and that the further from the pacemaker, the longer will be the compensatory pause. The pauses following auricular premature beats are never fully compensatory except in instances when the ectopic focus is situated very low—at the A-V node. In such a case, however, we are dealing in a strict sense with the nodal type of extrasystoles, which, in some instances at least, behave like ventricular extrasystoles in that they induce premature beats of the ventricles only and therefore are followed by complete compensatory pauses.

In Figure 41 the attempt is made to express diagrammatically the various possible compensatory pauses in relation to the various possible ectopic foci of impulse production in supraventricular extrasystoles. Such foci are naturally hypothetical and are chosen for the convenience of diagrammatic illustration.

FIG. 39.—Diagrammatic representation of the mechanism and extracardiac manifestations of Auricular Premature Beats (extrasystoles).

A, *Mechanism:* Rhythmic impulse production. The rhythm of the auricular response is disturbed in cycle 4 by an ectopic impulse arising in the lower portion of the auricles. It initiates a premature auricular contraction followed by a premature ventricular contraction: it spreads to the region of the S-A node and there annihilates an immature sinus impulse. The duration of the pause following the premature beat is somewhat longer than a normal intercycle period. Its length corresponds to the time it takes the ectopic impulse to reach the region of the normal pacemaker plus the time it takes to form a new impulse of the normal rhythmic series at the site of the normal pacemaker. The pause is not fully compensatory in that it does not make up for the brevity of the one preceding the premature beat. The ectopic auricular focus sets the pace for a single cardiac cycle.

B, *E. C. G.:* The fourth cycle is initiated by a *premature, inverted* P *wave* followed by premature ventricular complexes (Q, R, S, T) of normal configuration. The *pause* following the premature cardiac cycle is longer than any normal pause but is *not fully compensatory.*

C, *Heart Sounds:* In cycle 4, premature, somewhat feeble first and second heart sounds follow closely upon the normal sounds of the preceding cycle, duplicating them.

D, *Jugular Pulse:* Premature venous pulse (A, C) in cycle 4 followed by a pause.

E, *Carotid Pulse:* Premature, feeble arterial pulse beat in cycle 4 followed by a pause. The pulse beat following the pause is larger than any other normal pulse.

(For *mechanism* see also Fig. 41.)

AURICULAR PREMATURE BEATS. (EXTRASYSTOLES)

(A single premature beat arising in lower portion of auricles)

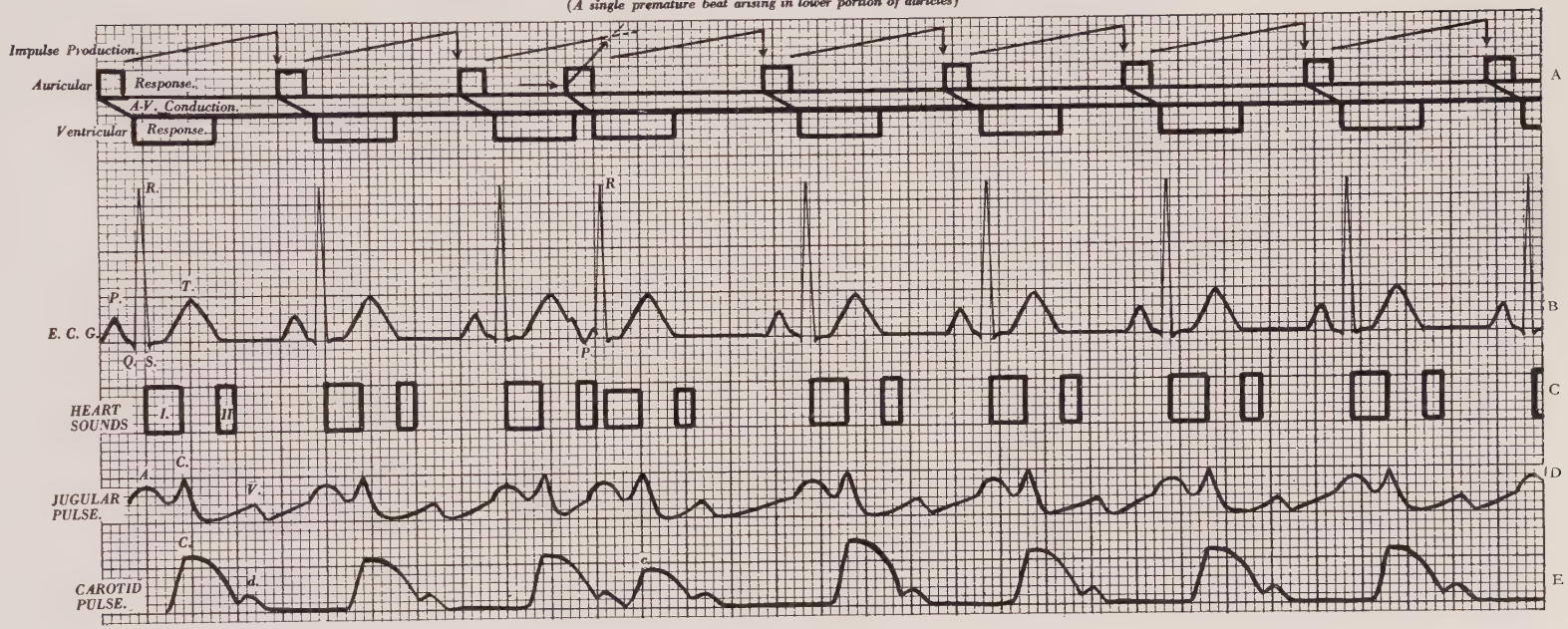

FIG. 39.

FIG. 40.—Diagrammatic representation of the mechanism and extracardiac manifestations of Auricular Premature Beats (extrasystoles—occurring frequently).

A, *Mechanism:* Cycles 3, 6 and 9 are initiated by an ectopic focus of impulse production situated in the lower portion of the auricle. The spreading ectopic impulse intercepts and annihilates the immature sinus impulses in the process of formation at the site of the normal cardiac pacemaker. The pause following a premature beat is longer than a normal cycle: it includes the time of the ectopic impulse spread and the time required to form a new sinus impulse. It is not fully compensatory. The ectopic focus sets the pace for every third cardiac cycle.

B, *E. C. G.:* Cycles 3, 6 and 9 are premature. The auricular complex P is premature and inverted; Q, R, S, T complexes are premature but of normal configuration. The pauses following the premature complexes are longer than the normal pauses but they are not fully compensatory.

C, *Heart Sounds:* Heart sounds of cycles 3, 6 and 9 are premature and feeble and they follow closely upon those preceding them (duplicating them). The sounds that follow the pauses are louder than the rest.

D, *Jugular Pulse:* Premature venous pulse groups in cycles 3, 6 and 9.

E, *Carotid Pulse:* Every third pulse beat is small and premature, giving the arterial pulse a trigeminal type of rhythm in which the first beat is the strongest and the third the weakest.

(For *mechanism* see also Fig. 41.)

AURICULAR PREMATURE BEATS. (EXTRASYSTOLES)

(Numerous premature beats arising in lower portion of auricles)

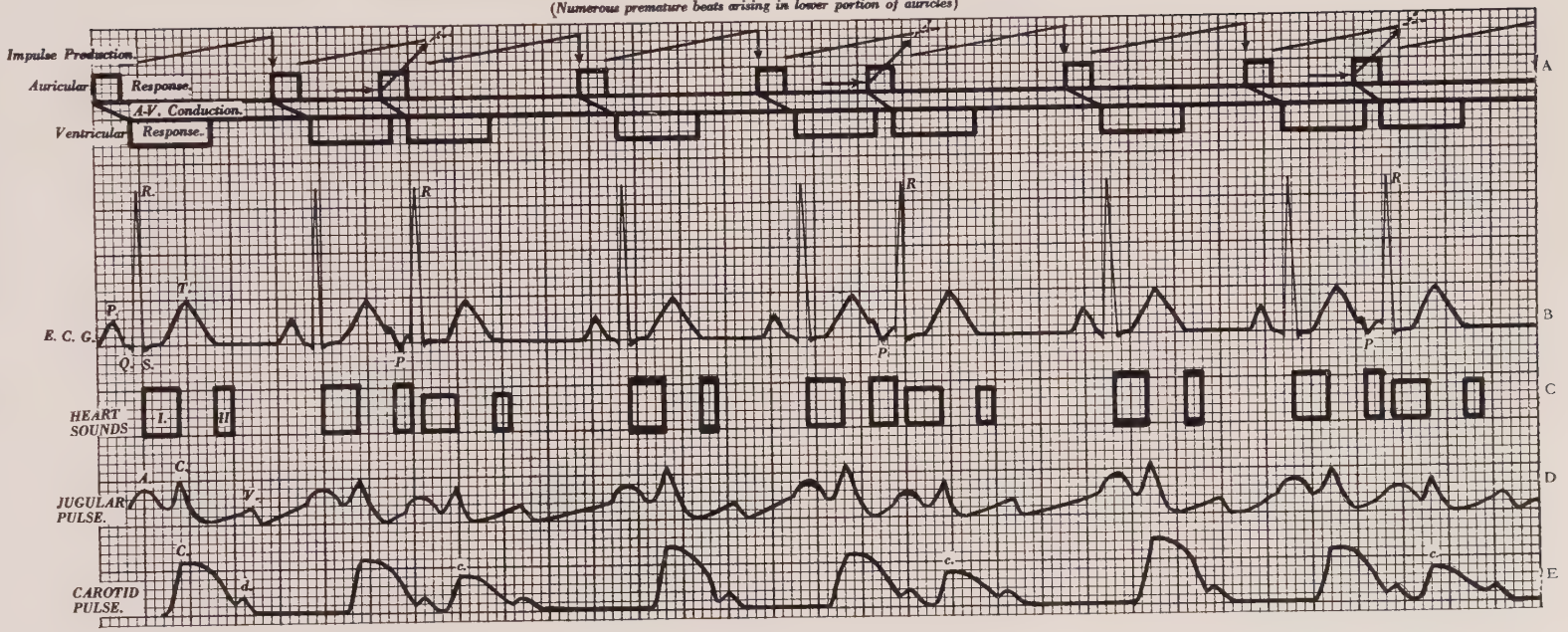

FIG. 40.

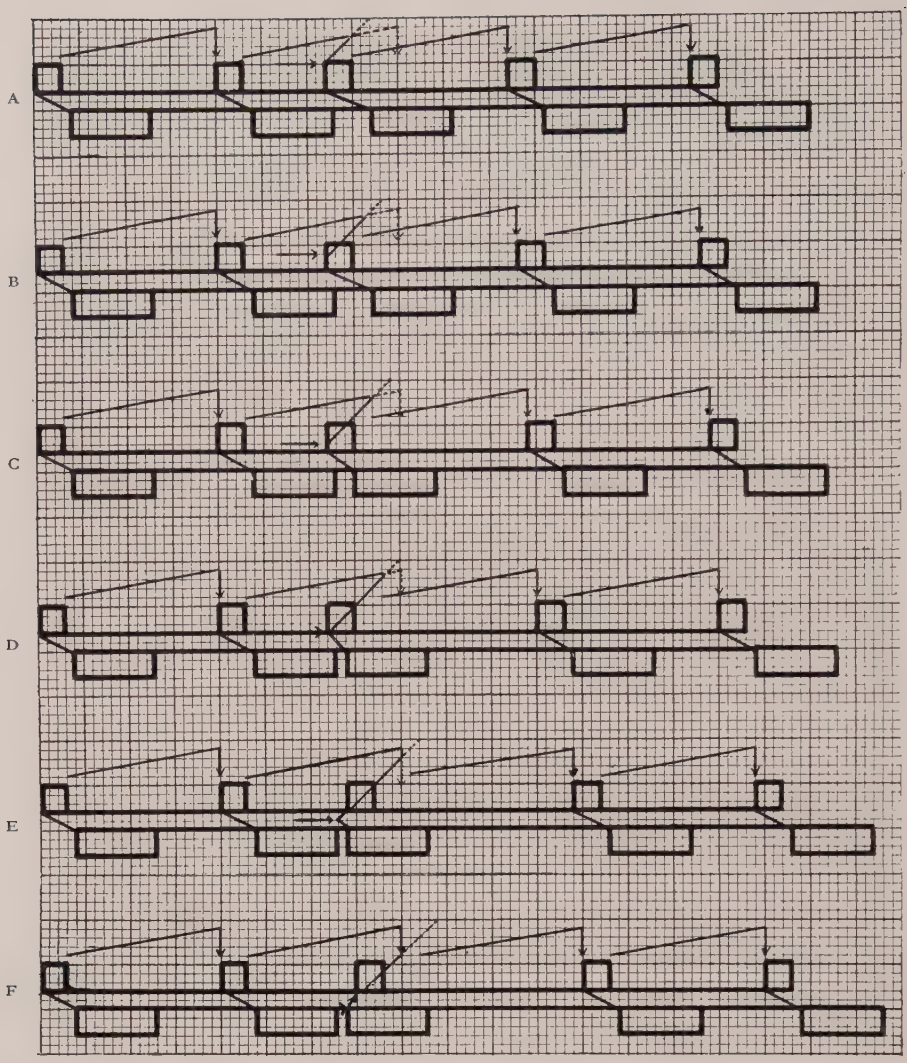

FIG. 41.—Diagrammatic representation of various supraventricular ectopic centers of impulse production and the manner in which they may determine the duration of the compensatory pauses succeeding them.[1]

A. In cycle 3, the arrow points to an ectopic center of impulse production situated in the upper part of the right auricle near the S-A node. A premature auricular contraction is induced ("sinus extrasystole"). The ectopic impulse arising in the immediate vicinity of the S-A node promptly extinguishes the immature sinus impulse of the rhythmic series. Following this, there begins the formation of a new sinus impulse at the usual rate of impulse production which, when complete, initiates the next normal rhythmic auricular response in cycle 4. Since there has been no appreciable loss of time on the part of the ectopic impulse

[1] *Diagonal lines* leading from point of ectopic impulse production (arrows preceding cycle 3) represent the *time* of impulse spread to the region of the S-A node.

in reaching the region of the S-A node and since the extinguishing of the immature sinus impulse is promptly followed by the formation of a new one at the normal sinus rate, the pause that follows the premature auricular beat is approximately equal to that which follows any normal beat in the same subject. The pause following a "sinus extrasystole" is not compensatory; in fact, its duration may even be shorter than that of a normal cycle.

B and C show the ectopic impulse arising in the middle and lower portions of the auricles respectively. In these cases, as in A, a premature auricular beat is induced, followed by a pause, which, in B and particularly in C, is definitely longer than a normal cardiac cycle. Since the ectopic focus is at some distance from the pacemaker proper, an appreciable time elapses before the ectopic impulse can extinguish the sinus impulse, so that the *beginning of the formation* of the new sinus impulse of the rhythmic series is slightly delayed, as shown in B, and moderately delayed, as seen in C. The pauses that follow the premature beats are somewhat compensatory in that they are longer than a normal pause. They represent the sum of a normal impulse production period plus the time required for the ectopic impulse to reach the site of the normal pacemaker. In C, the ectopic focus being at a lower level, the sinus impulse is intercepted later than in B, and therefore the pause following the extrasystole in C is greater than that in B; but *neither of them is fully compensatory*. A, B, and C represent the auricular type of supraventricular extrasystoles.

D, E and F show the ectopic focus to be situated in the vicinity of the A-V junctional tissues. In D, it is at the auricular portion of the A-V node; and in F at the ventricular portion of the A-V node or bundle of His. In these cases, the pauses following the premature beats are definitely prolonged. When the ectopic focus of impulse production is situated in the lowermost levels, as represented in E and especially in F, the pauses are often *fully compensatory;* i.e., the pause following the premature beats added to the one preceding it equals the sum of any two normal pauses. In this group, it is of interest to note that the relation and sequence between the auricular and ventricular contractions corresponding to the premature beats are also altered. For instance, in E the premature beats of auricles and ventricles are synchronous; in F the ventricular contraction actually precedes the auricular contraction. Broadly speaking, in each of these cases, the ectopic focus of impulse production is situated in the region of the junctional tissues, activating at the same time both the upper and the lower cardiac chambers. In D the auricles receive the stimulus just a short time before the ventricles. In E, they are stimulated synchronously; while in F the ventricles apparently receive the stimulus before the auricles. D, E and F represent the *"nodal" type of supraventricular extrasystoles.* The last form (F) resembles, in many respects, the ventricular premature beats described.

Clinical Features of Extrasystoles

Of all the arrhythmias, premature beats are most readily recognized, even by ordinary clinical means.

If, in the course of a rhythmic heart beat, there is a sudden duplication of the heart sounds followed by a pause and perhaps a sudden, feeble duplication of the arterial pulse, we are most likely dealing with extrasystoles.

THE HEART SOUNDS IN EXTRASYSTOLES

The heart sounds caused by premature beats may at times resemble the normal heart sounds of the subject; at other times, the character of such sounds (pitch, intensity, duration) may be profoundly modified. If the premature beat is effectual, that is, if it can raise sufficient intraventricular pressure to open the aortic semilunar valves, premature first and second heart sounds are heard, which differ from the normal in intensity only (they are generally weaker). If, on the other hand, the premature beat is ineffectual, that is, if it cannot open the semilunar valves, only a premature first heart sound is heard. Such a first sound may be markedly altered in character: it may be very short, loud and sharp (valvular in character) or, as occasionally happens, it may be very feeble and barely heard.

THE ARTERIAL PULSE IN EXTRASYSTOLES

The arterial pulse, whether it be coupled or not, also depends upon the ability of the premature beats to raise the semilunar valves. If effectual, the pulse is duplicated by a moderate or feeble premature pulse beat. If ineffectual, there is no coupling of the pulse at all: there is merely a long pause. *The pulse following in the wake of this pause,* the first of the normal rhythmic series, *is always larger than any other pulse beat,* because it follows a longer cardiac diastole and therefore a more complete diastolic filling of the heart.

THE JUGULAR PULSE

The jugular pulse is at times very characteristic. Visualization of the jugular vein, while the examiner at the same time auscultates at the apex, may at times show an unusually marked bulging which coincides with the premature heart sound at the apex. This bulging of the vein, as has been pointed out, is due to the fact that in some extrasystoles (ventricular) there is frequently a synchrony of contraction of both the upper and the lower cardiac chambers. The auricles, because of this synchrony, cannot discharge their contents into the contracting ventricles; the force of their contraction, therefore, is spent in intercepting the jugular onflow and in transmitting a pressure wave into the veins. In polygraphic tracings, the wave corresponding to the extrasystole is larger than any other single wave, because it is made up of two components, the auricular A wave and the ventricular C wave (Fig. 42).

EXTRASYSTOLES AND THEIR RELATION TO THE HEART RATE

Extrasystoles rarely accompany rapid heart action. They but rarely occur in hearts with rates of 100 or over. Premature beats manifest themselves in the predisposed generally when the heart rate is slowed, as during the relaxation following exercise. We often utilize this knowledge to induce extrasystoles for the purpose of study, as when, for instance, the heart appears regular but the clinical history suggests the occasional presence of extrasystoles; or when graphic records are desirable.

Often slight exercise followed by a short period of rest, a change of posture, slow deep breathing or the act of swallowing may suffice to induce them. On the other hand, acceleration of

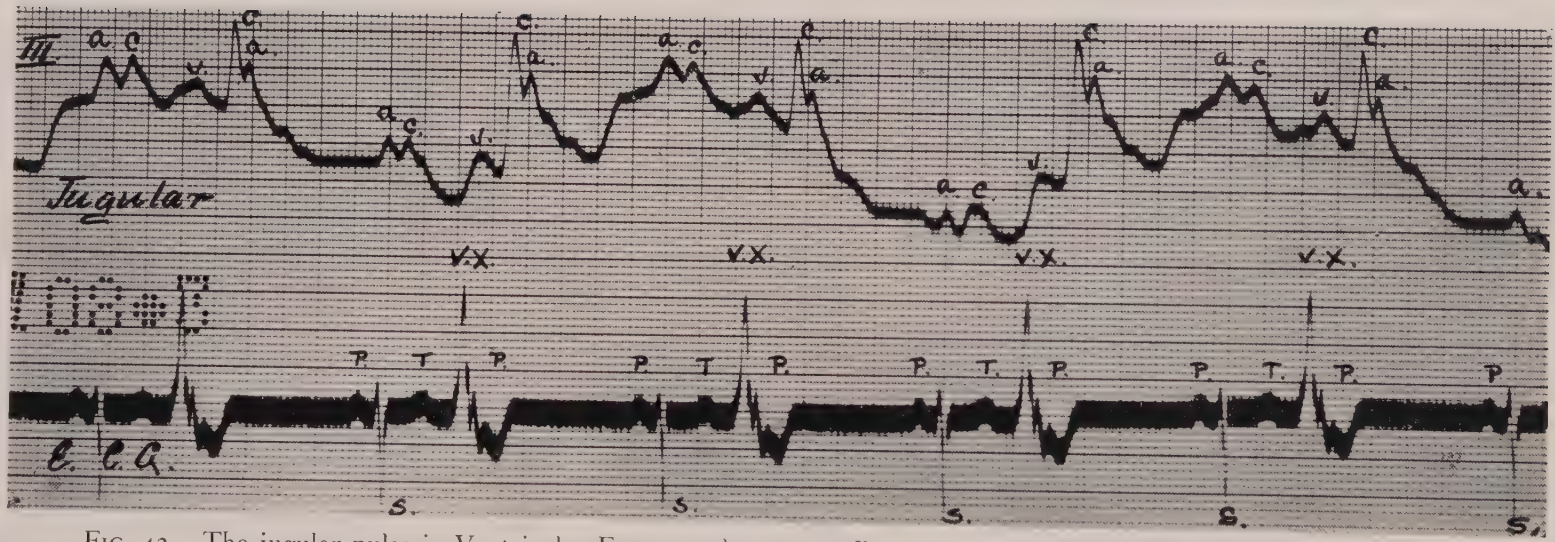

FIG. 42.—The jugular pulse in Ventricular Extrasystoles. Synchronous tracings of jugular pulse and electrocardiogram. The prominent deflection in the jugular pulse corresponding to the ventricular premature beat (vx) in the electrocardiogram, is the result of a summation of two elements, the ventricular C wave and the auricular A wave.

the heart rate from any cause tends to eliminate them. Exercise, fever, amyl nitrite or atropine may serve such a purpose.

CHARACTERISTIC ARRHYTHMIA OF EXTRASYSTOLES

When occurring in large numbers, extrasystoles often present a characteristic arrhythmia. Instead of a haphazard grouping which would manifest itself as a complex irregularity, they tend rather toward a regular recurrence; *an orderly grouping*. Premature beats may alternate with the normal cardiac cycles, in which case every second heart beat is an extrasystole (Fig. 43). If such grouping is present and if the extrasystoles are effectual (if they can force the semilunar valves), the resulting pulse is of a bigeminal type, that is, every alternate pulse beat is feeble—a "pseudo-alternans" (Fig. 44). If the extrasystoles which alternate with the normal beats are ineffectual (if they cannot open the semilunar valves), the pulse rate is "halved," namely the pulse is slow, regular and corresponds to half the actual rate of the heart beat (Fig. 45).

At times the grouping is such that, instead of alternating, an extrasystole may follow two normal cardiac cycles, so that only every third heart beat is premature. In such a grouping, if the extrasystole is effectual, there is a trigeminal rhythm to the pulse in which the first beat (the one induced by the normal heart beat following the pause) is the strongest and the third (the one induced by the premature beat) the weakest (Fig. 46). If, on the other hand, the extrasystole appearing every third beat is ineffectual, a slow pairing of the arterial pulse is noted (Fig. 47). Figure 48 shows a trigeminal arterial pulse due to ineffectual ventricular premature beats, appearing every fourth beat.

FIG. 43.—Diagrammatic representation of the mechanism and extracardiac manifestations of Ventricular Premature Beats (alternating with normal sinus rhythm).

A, *Mechanism:* Rhythmic impulse production: rhythmic auricular responses. Each alternate ventricular beat is premature and coincides with an auricular contraction. Having to wait for the next normal auricular impulse, each premature ventricular contraction is followed by a compensatory pause.

B, *E. C. G.:* Premature, bizarre ventricular complexes alternate with normal ventricular complexes. Every second normal P wave coincides with the terminal phase of a ventricular premature beat.

C, *Heart Sounds:* Coupling of the heart sounds: the first set of each couple is loud (cycles 3, 5 and 7 corresponding to the normal heart beat following a pause). The second set of each couple, on the other hand, is premature, sharp and of short duration (cycles 2, 4, 6 corresponding to the premature beats). Occasionally the second heart sound is barely heard —cycle 8.

D, *Jugular Pulse:* Alternate jugular pulses are prominent and have a single sharp crest resembling an arterial pulse beat (cycles 2, 4, 6 and 8, corresponding to the synchronous auricular and ventricular contractions).

E, *Carotid Pulse:* Bigeminal rhythm to the arterial pulse of which every second member is premature and feeble and is followed by a pause. The premature beat in the last cycle is ineffectual and does not induce a pulse.

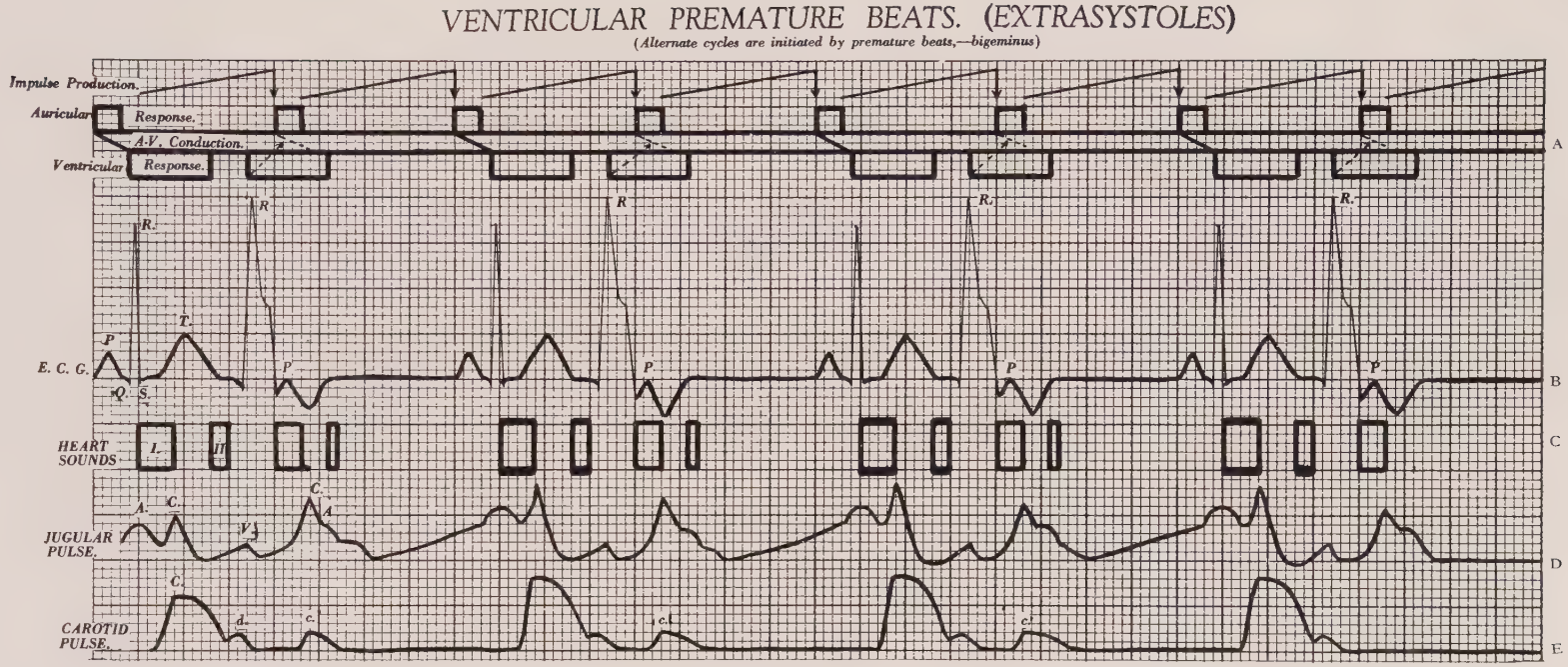
VENTRICULAR PREMATURE BEATS. (EXTRASYSTOLES)
(Alternate cycles are initiated by premature beats,—bigeminus)
Impulse Production.
Auricular
Response.
A-V. Conduction.
Ventricular
Response.
A
E. C. G.
P
T.
Q.
S.
R.
B
HEART SOUNDS
I.
II.
C
JUGULAR PULSE.
A.
C.
V.
D
CAROTID PULSE.
d.
c.
E

FIG. 43.

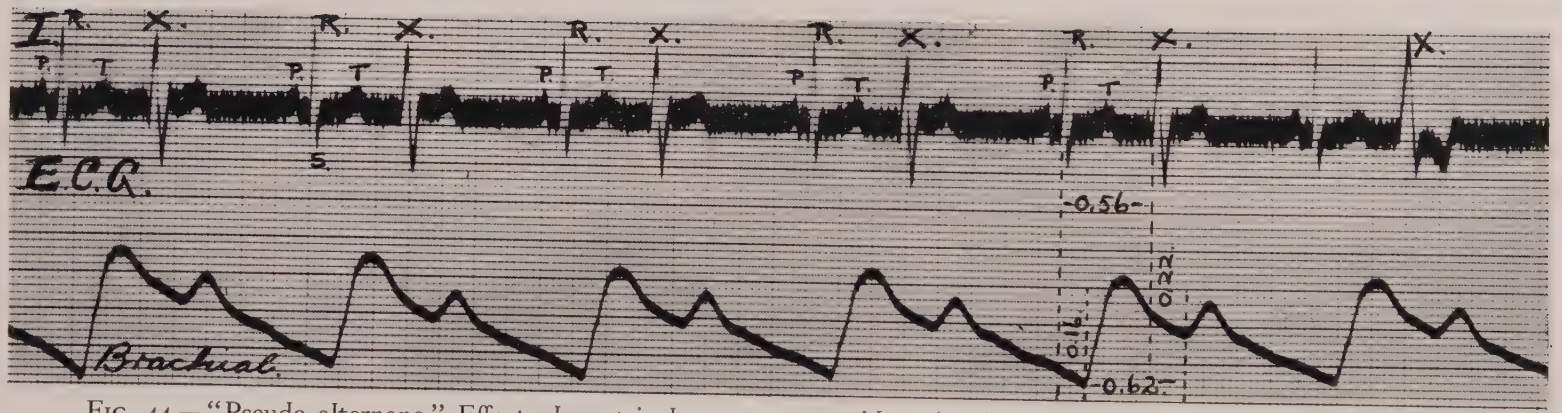

FIG. 44.—"Pseudo alternans." Effectual ventricular premature beats (x) appear every second cycle, inducing premature arterial pulses. Since transmission of the premature systole to the peripheral arteries takes longer than the systole of the normal heart beat (0.22:0.16) the prematurity is not as evident in the arterial pulse as it is in the electrocardiogram. Such a pulse, therefore, may clinically simulate a true pulsus alternans (equally spaced, alternating strong and weak pulse beats).

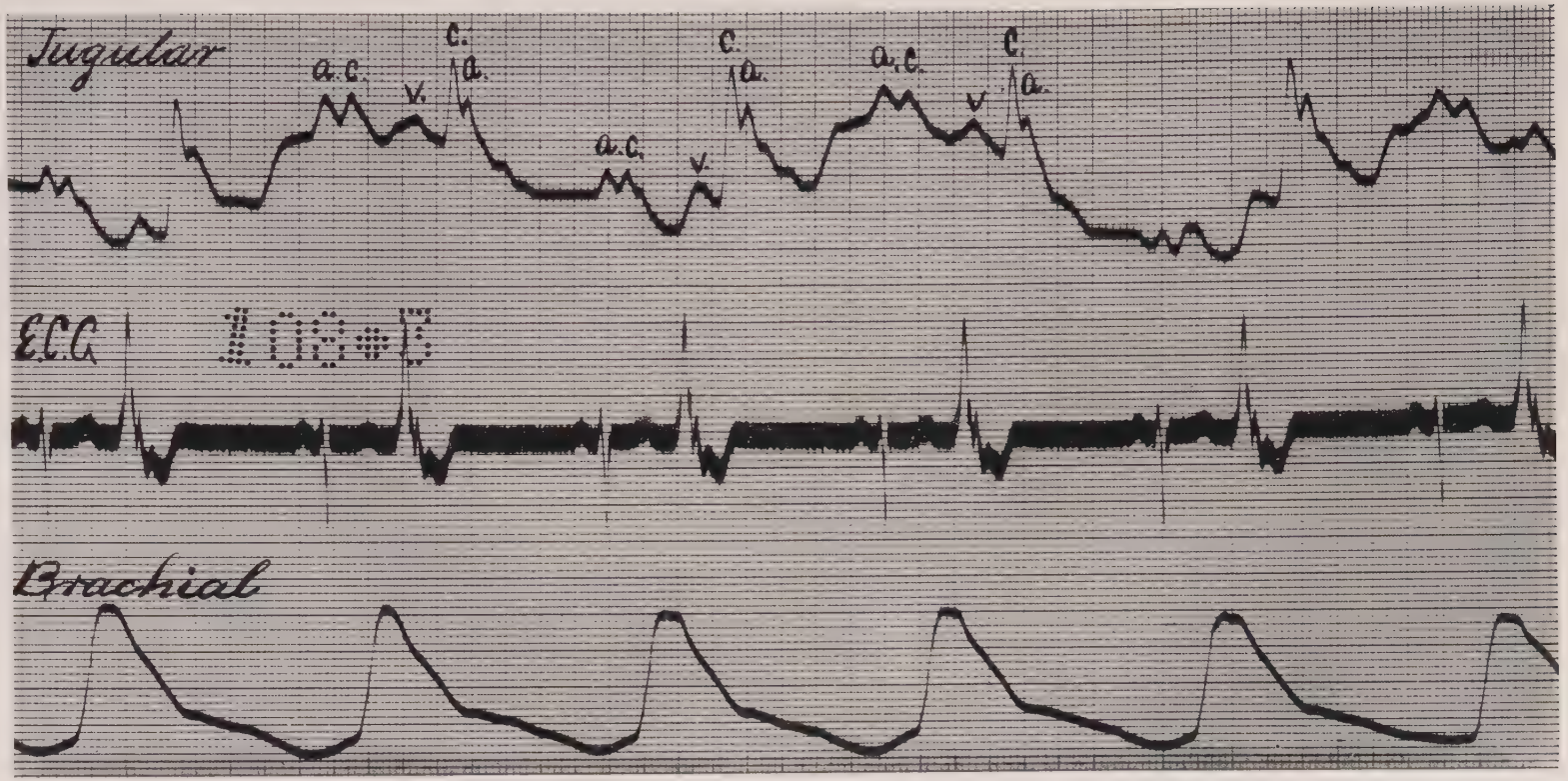

FIG. 45.—Halving of the arterial pulse. Venous pulse, electrocardiogram and arterial pulse, illustrating ineffectual ventricular premature beats. Every second beat is premature and fails to reach the peripheral arteries. This results in a "halving" of the arterial pulse. The jugular pulse corresponding to the extrasystole is prominent (synchronous contraction of upper and lower cardiac chambers).

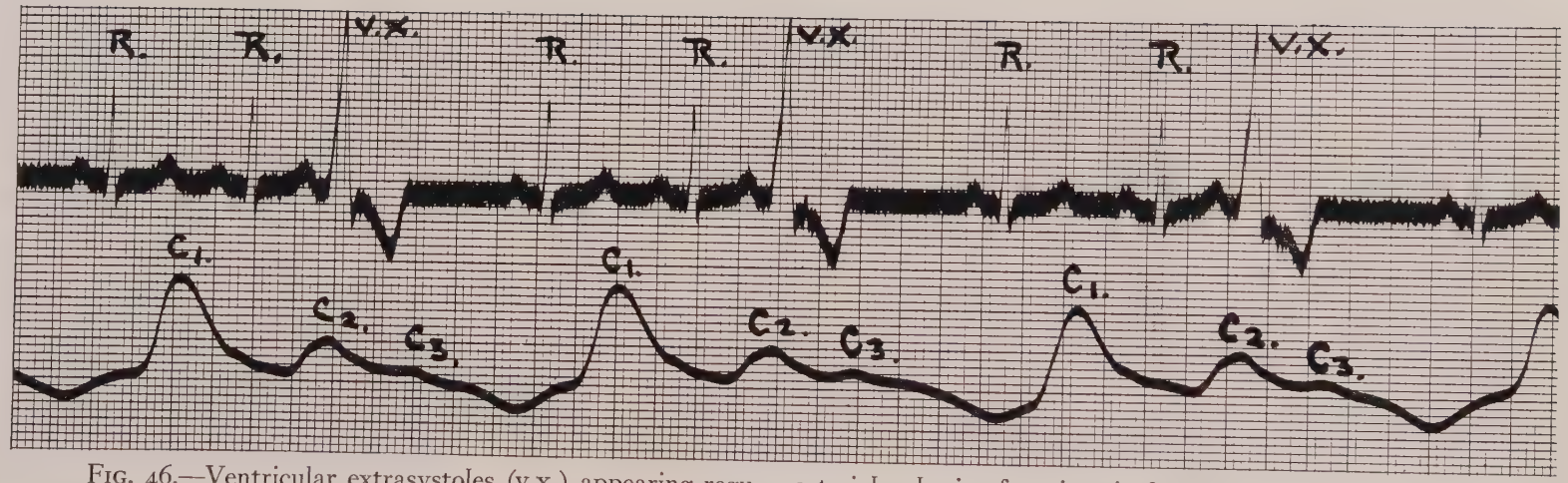

FIG. 46.—Ventricular extrasystoles (v.x.) appearing regularly every third beat. These premature beats are effectual in that they induce an arterial pulse. The grouping of the arterial pulse is of a trigeminal type in which the first of the group (following a pause) is the largest (c_1) and the last (due to the premature beat) is the smallest (c_3).

SUBJECTIVE SYMPTOMS OF EXTRASYSTOLES

The presence of extrasystoles may often be suggested by subjective symptoms on the part of the patient. Those who are predisposed to them, especially the thin-chested young and nervous type of patient, often complain of "palpitation." What such patients feel is, in fact, not a palpitation, as commonly understood, but rather a keen consciousness of an occasional abnormal heart beat. They often refer to a "peculiar sensation in the chest which feels as if the heart suddenly 'turned over' and then stopped." In many cases, however, it is not so much the premature abnormal beat as the first normal heart beat following the extrasystole that disturbs them. It is the unusually strong heart beat which follows the temporary standstill, the compensatory pause, that distresses them most. At times, the premature beat is associated with a tightness in the throat and often leads to considerable anxiety. The fact that extrasystoles tend to occur when the patient relaxes as, for instance, when first lying down to sleep or reclining after exercise, augments the mischief in those subject to this type of disturbance. The relaxation incident to retiring for the night often causes extrasystoles to appear in large numbers. The darkness and the perfect quiet of the sleeping room are highly conducive to the patient's consciousness of their presence. Great anxiety and often sleeplessness may follow.

CLINICAL DIFFERENTIATION BETWEEN AURICULAR AND VENTRICULAR PREMATURE BEATS

The clinical differentiation between auricular and ventricular premature beats generally requires graphic measures. There are, however, a few clinical hints which may serve as guides to their differentiation.

FIG. 47.—Pairing of the arterial pulse. Venous pulse, electrocardiogram and arterial pulse (brachial), illustrating ineffectual ventricular premature beats and the manner in which they manifest themselves in the venous and arterial pulses. Every third beat is premature and does not reach the peripheral arteries. This results in a "pairing" of the arterial pulse. The jugular pulse corresponding to the extrasystole is unusually prominent as a result of a synchronous contraction of upper and lower cardiac chambers. The venous curve also shows respiratory variations.

Fig. 47.

FIG. 48.—Trigeminal pulse. Venous pulse, electrocardiogram and arterial pulse (brachial) illustrating the influence of ineffectual ventricular premature beats on the venous and arterial pulses. Every fourth beat is an extrasystole which fails to reach the peripheral arteries; the arterial pulse, therefore, appears in groups of three. The jugular pulse corresponding to the extrasystole is unusually prominent as a result of a synchronous contraction of upper and lower cardiac chambers. The venous curve also shows respiratory variations.

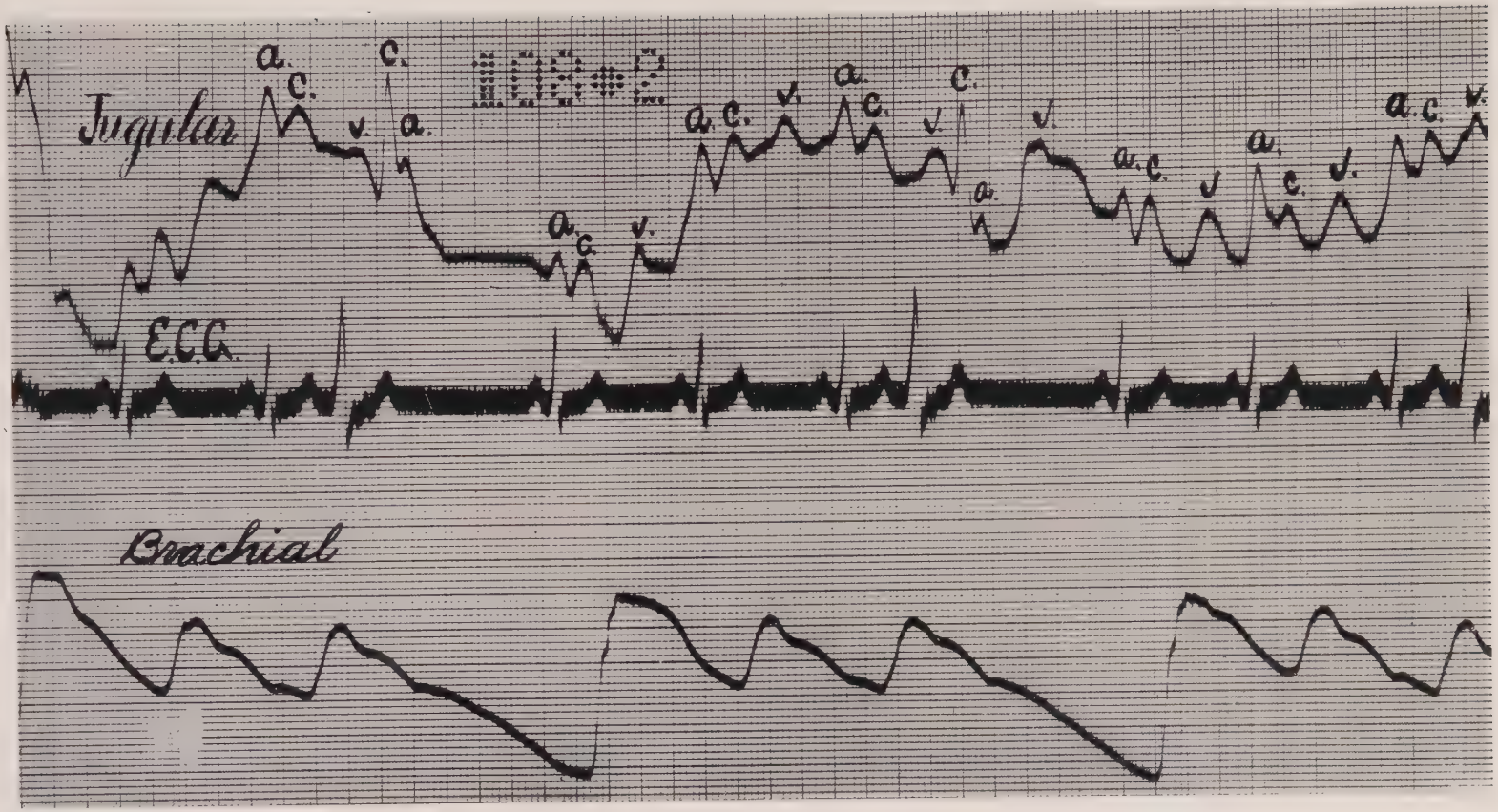

FIG. 48.

Ventricular premature beats are frequently encountered in the middle aged. In fact, in these individuals it is a very common cause of a cardiac arrhythmia. Extrasystoles, when accompanying or following intensive digitalis medication, are generally also of ventricular origin.

Because of the short diastoles that precede them and the imperfect filling of the ventricular cavities (lack of previous auricular contraction), ventricular extrasystoles often fail to raise the aortic valves. Therefore, in these cases, as a rule, only the first heart sound is duplicated. The character of this first heart sound is often markedly altered, manifesting itself to the listener, at times, as a sudden, sharp slap; at other times, as an extremely feeble sound. For similar reasons the arterial pulse, in ventricular premature beats, is also less apt to be duplicated so that a long pause may be the only arterial manifestation. The jugular pulse corresponding to a ventricular premature beat is often of huge amplitude. Ventricular extrasystoles, often being of the ineffectual type, perform but little work in propelling the arterial blood.

Auricular premature beats, on the other hand, are more apt to be effectual because a premature auricular contraction precedes and, in fact, is the cause of each premature ventricular contraction; also because the diastoles preceding such premature heart beats are generally somewhat longer than those found preceding ventricular extrasystoles. Consequently, there is better filling of the ventricles and the subsequent premature beat is more likely to raise the semilunar valves. Because of this, the heart sounds (first and second) are usually coupled in the case of auricular premature beats and the character of the premature heart sounds differ but slightly from the normal sounds immediately preceding them. The arterial

pulse is also generally duplicated in the case of auricular premature beats.

INCIDENCE OF IRREGULAR HEART ACTION IN RELATION TO PREMATURE BEATS

Irregular heart action as a result of premature beats is a very common disorder. To be sure, statistics dealing with the relative frequency of the various arrhythmias do not give it first place; such statistics, however, gathered in hospitals where the more acute forms of cardiac disorders predominate, are wholly unreliable. The great majority of those having extrasystoles do not go to hospitals; many, in fact, are not at all aware of the presence of the disorder.

ETIOLOGY OF EXTRASYSTOLES

Our views regarding the etiology of extrasystoles are based largely on speculation. Extrasystoles are occasionally met with in young children[1]; most often, however, they are found in the middle-aged. Almost every one has them at one time or other. They are not necessarily associated with grave cardiac conditions; in fact, they are rather infrequently met with in the course of decompensation as a result of valvular heart disease or myocardial involvement due to coronary artery disease, except perhaps at the time of a sudden occlusion of one of these vessels. To assume a pathological condition, therefore, every time we encounter extrasystoles, especially in the face of the great frequency with which they occur in the apparently healthy, would be giving these disorders an entirely unjustifiable and undue prominence.

[1] Bass, M. H. The significance of cardiac extrasystoles in childhood. *J. Am. M. Ass.* 1926, lxxxvi, 387.

No doubt, temporary *disturbances in cardiac nutrition* play an important rôle in some cases; while in others, *toxic states* of the myocardium may be the underlying causes. *Certain drugs*, notably those of the digitalis group and quinidine, may cause extrasystoles to appear in large numbers, together with other evidences of toxic involvement of the specific system and the myocardium proper (sino-auricular block and auriculo-ventricular heart block). Accordingly when extrasystoles appear, the further use of these drugs is contraindicated. We have no means of judging the possible effects of *endogenous toxins* (gastrointestinal, uremic, etc.) upon the intrinsic cardiac mechanism. They, too, may play a part. It is of interest to note that extrasystoles have been observed in the presence of *focal infections* and that the eradication of the infection was promptly followed by the termination of the extrasystolic arrhythmia.[1] *Nervous influences* are known to enhance the frequency with which extrasystoles may appear; but it is questionable whether nervous impulses can ever induce them in a perfectly healthy organ. It is more likely that the nervous influence merely augments the mischief in a heart that is already the seat of some pathological process or one that bears the brunt of at least a mild toxemia.

In any event, judging from the foregoing, the *mere presence* of extrasystoles cannot be taken as evidence of heart disease and cannot be used as a guide in prognosis. To quote Lewis: "They serve a purpose in diagnosis by directing attention to the heart, but are of no value in prognosis."[2]

[1] Smith, S. C. Dental infections and heart irregularities. *M. J. & Record* (suppl.), 1925, cxxii, 207–310.

[2] Lewis, T. Clinical Disorders of the Heart Beat. Lond., 1921, p. 56.

MANAGEMENT OF PATIENTS WITH EXTRASYSTOLES

Having ruled out organic heart disease, the management of those subject to extrasystoles resolves itself into the application of general measures, some of which are employed for relief and others with the hope of preventing recurrences. There is no specific drug known to influence the arrhythmia. Digitalis as a direct remedy is contraindicated. Hypnotics in appropriate doses often serve to allay the patient's anxiety and may even remove a probable etiological (nervous) factor. Reassurance may have a similar effect upon a very anxious and perturbed patient. As a prophylactic measure, the removal, as far as possible, of all exogenous and endogenous toxins such as tobacco, drugs of the digitalis-strophantus group, gastro-intestinal toxins, etc., is indicated. A diligent search for foci of chronic infections and their prompt eradication may strike at the root of the disorder.

CLINICAL FEATURES OF THE EXTRASYSTOLIC ARRHYTHMIAS

	Hemodynamics during Slow Phases			SPECIAL FEATURES
	Heart Sounds	Jugular Pulse	Arterial Pulse	
Ventricular premature beats	Rarely premature 1st and 2nd. Usually sharp premature 1st or faint premature 1st only.	Prominent, especially if heart is slow.	Faint premature pulse or pause only.	Slow phase initiated by coupling of heart sounds, occasionally by coupling of pulse. When numerous, they give rise to bigeminal rhythm, trigeminal rhythm, halving or pairing of the arterial pulse: occasionally to paroxysms of tachycardia. Subjective sensations are often suggestive. They occur in the course of digitalis or quinidine medication, mild toxic states of the heart muscle, focal infection and sclerotic heart disease. They at times complicate other arrhythmias.
Auricular premature beats	Usually premature 1st and 2nd. Rarely premature 1st.	May be prominent.	Premature pulse usually present. Rarely pause only.	

These arrhythmias tend to subside, at times even to disappear, on acceleration of the heart rate from any cause: exercise, fever, amyl nitrite, atropine, etc.

They tend to be augmented by vagotropic drugs or toxins: digitalis, pilocarpine, morphine, etc.

Chapter IX

Paroxysmal Tachycardia

RAPID heart action is a common clinical phenomenon and is observed under various conditions. Infections or intoxications are generally associated with moderate or marked tachycardias, lasting for varying lengths of time. Even in normal individuals, short periods of rapid heart action are readily induced by comparatively trivial causes, such as exercise or emotional states. In these so-called *simple tachycardias* the onset as well as the termination is brought on *gradually* and the degree to which such rapid heart action rises depends, at least in part, upon the severity of the exciting factor.

Paroxysmal tachycardia, on the other hand, is a specific type of disorder of the cardiac mechanism which manifests itself clinically as a periodic, *abrupt* acceleration of the heart beat. In this condition, as in simple tachycardias, the essential disturbance in the heart is a disturbance of rate and not of rhythm. Nevertheless, paroxysmal tachycardia is included in this treatise on cardiac arrhythmias because it is regarded as an occasional clinical manifestation in a group of disorders of the cardiac mechanism, the milder types of which usually appear as irregularities of the heart beat (extrasystoles).

Mechanism of Paroxysmal Tachycardia

In the chapter on premature beats (p. 109), it was observed that while normally the cardiac pacemaker dominates the cardiac rate and rhythm, occasionally a premature impulse, elaborated in an ectopic focus or secondary center of impulse production, may initiate a heart beat. If the premature stimu-

lus is supraventricular, that is, auricular or "nodal" in origin, the cardiac response is designated as a supraventricular extrasystole and the resulting premature contraction involves the whole heart: auricles and ventricles. On the other hand, if the premature impulse arises in the ventricles, the cardiac response is designated as a ventricular extrasystole and the resulting premature cardiac contraction generally involves the ventricles alone. In any event, the *ectopic focus* that gives rise to a premature heart beat *becomes the temporary pacemaker* of the heart for at least a single cardiac cycle.

Premature beats, it was observed, may at times appear with considerable frequency. When numerous, they may alternate with the normal heart beats, in which case the ectopic focus or subsidiary center alternates with the normal cardiac pacemaker in setting the pace for the heart. Since extrasystoles may at times occur in pairs, the ectopic focus in such a case sets the pace for two successive cardiac cycles.

The irritability of an ectopic focus of impulse production may at times become so heightened that it will set the pace for *a series of premature heart beats*, subduing, for the time being, the normal cardiac pacemaker. Such series of premature beats manifest themselves clinically as an abrupt acceleration of the heart rate for a group of beats. This constitutes *a paroxysm of tachycardia*. The duration of a paroxysm is very variable from individual to individual, though it is fairly constant in a given subject. Some paroxysms may last but a few seconds or minutes, while others may continue for hours or even days. The very short paroxysms generally precede the onset or immediately follow upon the termination of a long-continued paroxysm.

As in the case of extrasystoles, so in paroxysmal tachycardias, the type is determined by, and designated according

to, the site of origin of the ectopic impulse. Accordingly, we recognize an *auricular paroxysmal tachycardia*, a *"nodal" paroxysmal tachycardia*, (both of supraventricular origin) and a *ventricular paroxysmal tachycardia.*

No matter where the site of the ectopic focus of impulse production may be, so long as it maintains its temporary rôle of pacemaker, such a focus, for the time being, dominates the situation absolutely. It sets a rapid pace for the heart and keeps the normal cardiac pacemaker in abeyance. The short diastoles incident to the rapid heart action do not permit the production of mature impulses at the site of the normal cardiac pacemaker. The sino-auricular node probably does elaborate stimuli successively, but can build them only to a partial stage of maturity, because the rapidly recurring impulses, emanating from the dominant ectopic focus, probably intercept and thus extinguish each of them successively while they are still in a state of immaturity.

This is particularly true in the case of paroxysmal tachycardias of supraventricular (especially auricular) origin. In these types, the impulses arising in the ectopic focus spread primarily over the auricular musculature and therefore readily involve also the region of the sino-auricular node. Figure 49 is a diagrammatic representation of a paroxysmal auricular tachycardia and depicts such a mechanism. It shows that the cardiac pacemaker does elaborate impulses successively, but that each of them in turn, while still immature, is intercepted and extinguished by the rapidly recurring premature impulses emanating from a dominant ectopic focus, situated in the lower portion of the auricular musculature. It represents a short paroxysm of tachycardia showing an abrupt onset and termination as a result of the abrupt changes in the cardiac mechanism.

FIG. 49.—Diagrammatic representation of the mechanism and extracardiac manifestations of Paroxysmal Auricular Tachycardia.

A, *Mechanism:* Beginning with cycle 3, the ectopic focus of impulse production, situated in the lower portion of the auricle, succeeds in setting the pace for eight cardiac cycles at a rapid rate. Each successive ectopic stimulus intercepts an immature sinus impulse, keeping the normal cardiac pacemaker in abeyance for the time being. Following the paroxysm, there is a pause which is not fully compensatory. Because of a sudden change in the intrinsic cardiac mechanism, in cycles 3 and 10, the paroxysm begins and terminates abruptly.

B, *E. C. G.:* A series of premature, inverted P waves followed by premature ventricular complexes of normal configuration constituting a paroxysm.

C, *Heart Sounds:* A sudden onset of a series of rapidly recurring 1st and 2nd heart sounds of a "tic-tac" quality terminating suddenly.

D, *Jugular Pulse:* A series of feeble venous pulse waves.

E, *Carotid Pulse:* A sudden onset of a series of small rapid arterial pulse beats, beginning abruptly and terminating just as abruptly.

PAROXYSMAL AURICULAR TACHYCARDIA.

(*A series of auricular premature beats*)

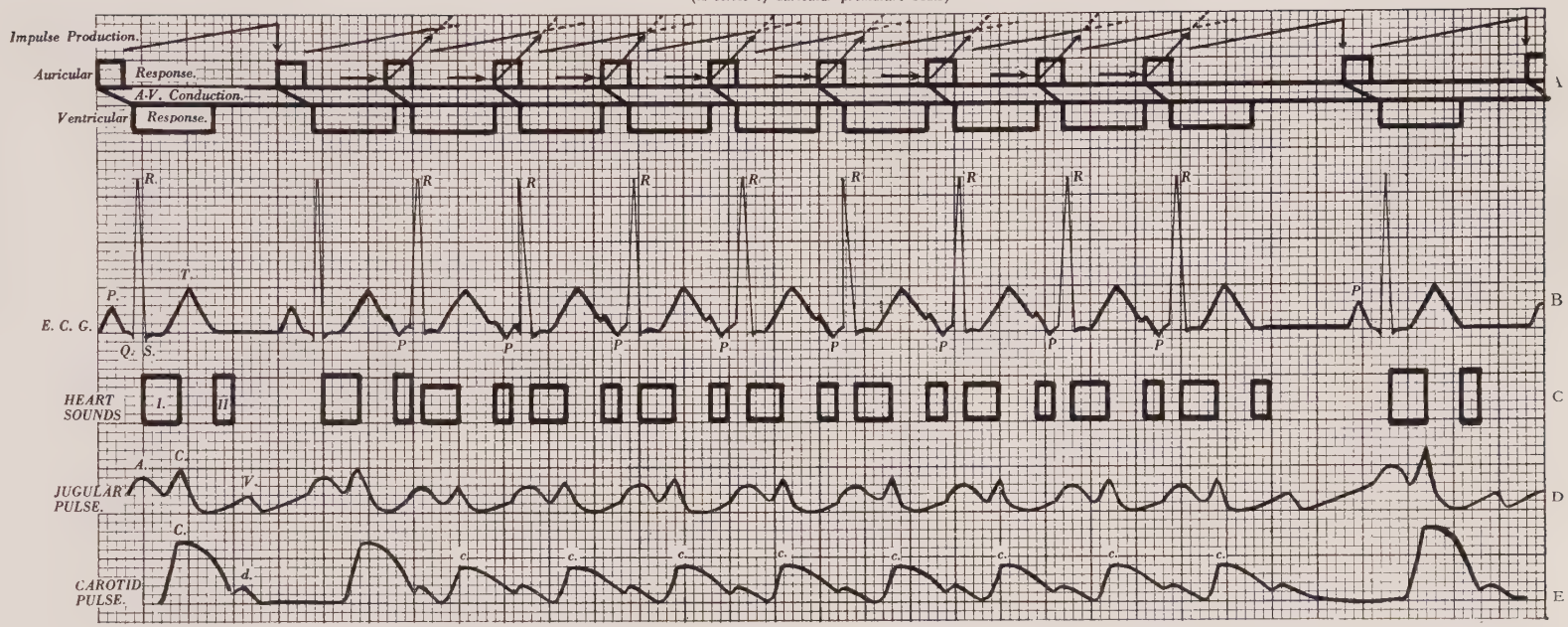

FIG. 49.

In paroxysmal tachycardia of ventricular origin (Fig. 50), impulses from the temporary pacemaker do not, as a rule, interfere with the normal cardiac pacemaker. The site of impulse production being in the ventricle, the premature impulses, in order to interfere with the pacemaker, would have to spread over a greater area, i.e., travel a greater distance and traverse bizarre pathways. The impulse in such case, in order to interfere with the normal pacemaker, would have to bridge the relatively refractory junctional tissues in a retrograde direction. This is generally impossible. Therefore, in a paroxysmal tachycardia of ventricular origin, while the ventricles beat rapidly in response to an ectopic focus within their own substance, the auricles generally respond to the normal rhythmic series of stimuli that arise at the site of the normal cardiac pacemaker, the sino-auricular node. The auricular rate is, therefore, generally normal and independent. Occasionally, a ventricular impulse, one of the ectopic and rapid series, may succeed in bridging the junctional tissues and induce a retrograde auricular response; thus it may at times upset the normal, independent auricular rhythm (Fig. 38, cycle 8).

Clinical Features of Paroxysmal Tachycardia

Since the cardiac rhythm in paroxysmal tachycardias is undisturbed, an attempt at correlation of their extracardiac manifestations would aid but little in their recognition.

The heart rate is rapid, 150 to 200 a minute. The heart sounds often assume the embryonic type of "tic-tac" quality.

THE ARTERIAL AND JUGULAR PULSES

The arterial pulse is rapid, feeble and generally regular. Graphic records often show it to be a "pulsus alternans"; that

is, larger and smaller pulse beats alternate with one another. Because of its rapidity and feebleness, the pulse at times seems irregular. It is often difficult to count the pulse beats at the wrist, and one must resort to the apex impulse for an accurate estimation of the heart rate.

The jugular veins are generally engorged and pulsate feebly; at times, the pulsations may be definitely seen or even felt over them. The venous pulse is of no diagnostic value.

ELECTROCARDIOGRAPHIC FINDINGS

An electrocardiogram taken during the attack may not always disclose the true nature of the disturbance. It may simply show a tachycardia in which the diastolic intervals are markedly reduced, at times almost to zero. In supraventricular tachycardias the approximate location of the ectopic focus of impulse production is at times definitely indicated by an inversion of the auricular P wave. All other complexes however, are generally of normal configuration (Fig. 49).

In ventricular paroxysmal tachycardias, the electrocardiogram generally shows bizarre ventricular complexes in which the initial deflections are widened (see form of ventricular extrasystoles). Definite auricular P waves are not seen, as a rule. When they accidentally chance to coincide with some of the ventricular complexes distorted forms may be recognized. Since there is no definite time relation between the auricular and ventricular contractions, the P wave may fall into any part of the ventricular phase and thus may at times alter the initial deflection, and at other times the final deflection of the ventricular group of complexes (See E.C.G. in Figure 50).

Rarely the ectopic ventricular focus of impulse production may succeed in influencing the auricular rate. The junctional

FIG. 50.—Diagrammatic representation of the mechanism and extracardiac manifestations of Paroxysmal Ventricular Tachycardia.

A, *Mechanism:* Rhythmic impulse production: rhythmic auricular responses. Beginning with cycle 3, there is an abrupt onset of a series of premature ventricular beats which terminate abruptly with cycle 10. These constitute a short *paroxysm of tachycardia.* The ventricular rate during the paroxysm is rapid, more than twice the rate of the auricles which beat in response to the normal sinus impulses. Auricular and ventricular contractions frequently coincide.

B, *E. C. G.:* Beginning with cycle 3, there is a series of closely spaced, bizarre ventricular complexes ending with cycle 10. The normal rhythmic P waves break in upon these ventricular complexes with a constantly varying relation to them.

C, *Heart Sounds:* A sudden change in the rhythm and character of the heart sounds beginning with cycle 3 and ending with cycle 10.

D, *Jugular Pulse:* A series of intricate, feeble indulations of venous pulse beats for ten cycles. (It serves no particular diagnostic purpose.)

E, *Carotid Pulse:* A sudden change in the rate and volume of the arterial pulse beginning with cycle 3 and terminating with equal abruptness in cycle 10.

PAROXYSMAL VENTRICULAR TACHYCARDIA.

(A series of ventricular premature beats)

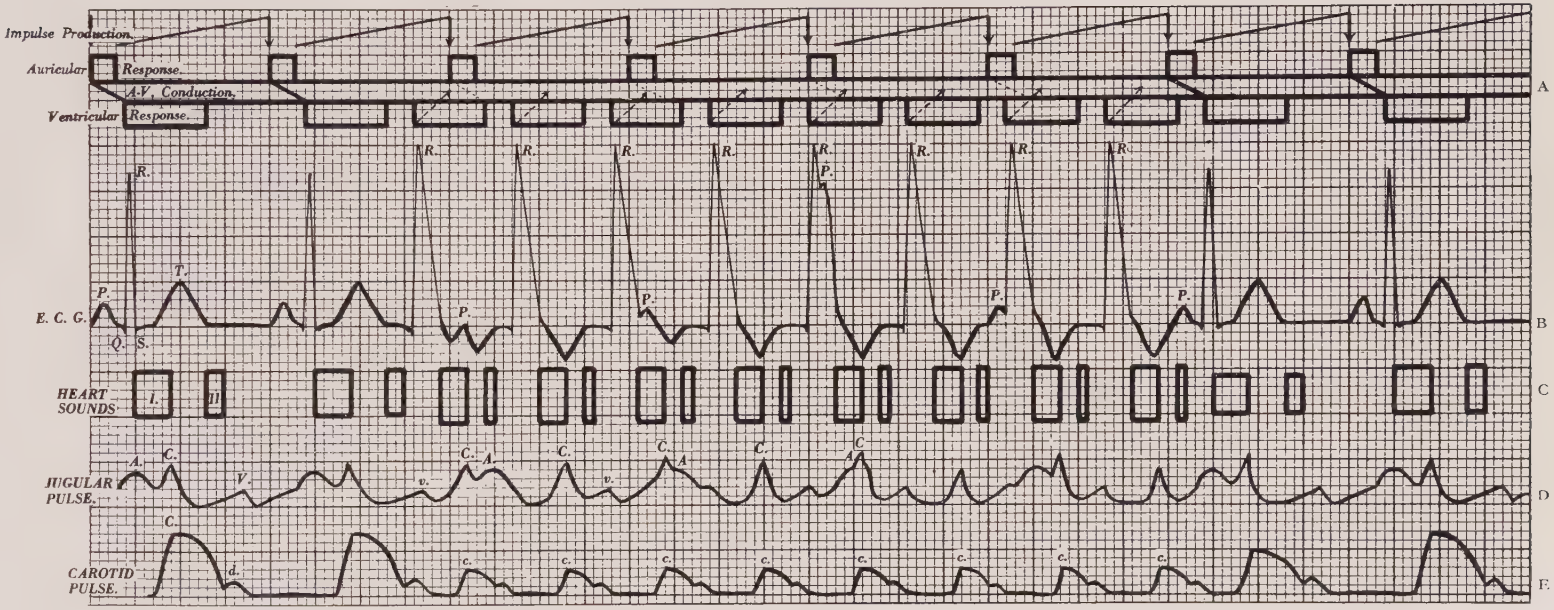

FIG. 50.

tissues permitting, such ectopic focus may send every second or every third impulse to the auricles, thus determining the rhythm of the upper chambers. Since all the retrograde impulses cannot reach the auricles, since some of them are blocked, this phase of the disturbed cardiac mechanism is referred to as a reversed A-V block or a ventriculo-auricular heart block.

Electrocardiographic tracings taken during the intervals, between attacks, may at times suggest the type of the paroxysmal tachycardia. Patients subject to paroxysmal tachycardia often have, during the free intervals, isolated or occasionally grouped extrasystoles. This is particularly true shortly before or shortly after an attack. The extrasystoles recorded during such interval periods denote the presence of an active ectopic focus. The type of extrasystole (supraventricular or ventricular) suggests the location of the ectopic focus, and, since the configurations of the complexes of the interval extrasystoles generally conform to the complexes seen during a paroxysm in the same subject, the type of these interval extrasystoles serves to identify the type of the tachycardia which constitutes the paroxysm.

CLINICAL HISTORY AND APPEARANCE OF THE PATIENT

The clinical appearance of the patient during an attack, together with a history of previous similar attacks, often discloses the nature of the malady. A history of previous attacks of rapid heart action which *came on abruptly and stopped just as abruptly* and which lasted but a few hours, when elicited in the case of a patient who is suddenly taken with severe palpitation, shortness of breath, with perhaps some tenderness over the region of the liver and moist râles at the lung bases, points very strongly to

the nature of the disorder as being a case of paroxysmal tachycardia.

The fact that change of posture or mild exertion has no appreciable effect upon such rapid heart action serves as an additional clinical evidence.

Patients with paroxysmal tachycardia, except for the consciousness of a palpitation and the associated anxiety, often present a clinical picture of comparative well-being, rarely seen in any other type of rapid heart action. In fact, some are not even aware of the presence of an attack of short duration. Signs of heart failure are generally absent during the early stage of an attack.

The consciousness of a sudden onset and of a persistent palpitation disturbs these patients more than the actual circulatory embarrassment.

If the attacks last many hours or perhaps days, or if the patient is of middle age (in such a person the myocardial reserve is limited), signs of heart failure, such as marked dyspnea, engorged liver, edema of the extremities or ascites, gradually supervene. Rarely pulmonary edema sets in, and unless the attack terminates quickly, it leads to rapid, fatal circulatory failure.

Troublesome symptoms, such as precordial pain or pain over the engorged liver, occasionally arise. Patients showing such symptoms, when seen in an attack for the first time, may offer some difficulty in differential diagnosis. Because of rapid heart action, dyspnea and moist râles in the lungs, the clinical picture may simulate an acute pulmonary disease. On the other hand, if epigastric pain and vomiting, as a result of hepatic congestion, are the predominating symptoms, the condition may be mistaken for an acute intraabdominal disease.

Attacks of paroxysmal tachycardia generally stop spontaneously. At times, however, one can elicit the interesting history that patients subject to these disorders can stop their own attacks. This they accomplish by a variety of measures, such as breath-holding, assuming a crouching position or the knee-chest posture.

Pressure over the right carotid artery in the neck or pressure over the eyeballs may at times be employed to stop some paroxysms, at least temporarily. This is generally true in cases of auricular paroxysmal tachycardias in young people.

Chapter X

Auricular Flutter and Auricular Fibrillation

AURICULAR flutter and fibrillation comprise a group of clinical disorders of the heart beat in which the intrinsic auricular mechanism is profoundly altered. The two conditions are described under one general heading, because, although distinct and separate clinical entities, they are, nevertheless, fundamentally probably similar in that they represent two types of manifestation of the same peculiar disturbance of the intrinsic cardiac mechanism. Clinically, auricular flutter generally appears as a regular and often also as a rapid heart action, in which case it resembles in many respects a paroxysmal tachycardia. Occasionally, however, it may present the appearance of a complete irregularity. Auricular fibrillation, on the other hand, almost always manifests itself clinically as an absolute irregularity of the heart beat.[1]

Significant Historical Facts

Auricular flutter and auricular fibrillation have an interesting historical background. Clinical observations on *auricular flutter* date back to the observation of Hertz and Goodhart in 1908 who first drew attention to this condition as a clinical disturbance in the human heart.[2] Jolly and Ritchie in

[1] Exceptions. 1. If there is an associated auriculo-ventricular heart block, the ventricles may beat regularly, because in such a case, the ventricles respond to an independent source of impulse production and are not at all influenced by stimuli coming from the upper chambers. 2. Dock, W., and Levine, S. A. The occurrence of regular ventricular rhythm with a rate of over fifty in cases of auricular fibrillation. *Am. J. M. Sc.*, 1924, clxvii, 664.

[2] Hertz, A. F., and Goodhart, G. W. The speed-limit of the human heart. *Quart J. Med.*, Oxford, 1908–9, ii, 211–218. (P. 474, ref. 370.) (Figures in parentheses refer to the pages and reference numbers in Lewis, T. "Mechanism and Graphic Registration of the

1910[1] and Ritchie in 1912–13–14[2] were also among the early contributors. The term flutter was employed, though vaguely applied, as far back as 1887.[3]

These early observations brought out many of the physiological and clinical features which characterize auricular flutter. They revealed the interesting facts that in flutter the auricles contract very rapidly and with a remarkable constancy, and that the ventricular response may vary within a wide range; namely, that the response may be rapid or slow and either perfectly regular or markedly irregular.

The actual mechanism of the underlying disturbance in clinical flutter was practically unknown prior to 1920. Hypotheses were put forward, to be sure, the most attractive of which was that flutter arose abruptly as a new rhythm in an ectopic focus. Evidence was even offered to suggest that the most probable origin of this new and very rapid rhythm was in the S-A node.

Auricular fibrillation was long recognized as a condition in which the auricles were dynamically inactive in so far as they showed no evidence of gross contraction and that they were practically ineffectual in propelling the blood. The ventricles, on the other hand, were known to beat with an absolutely irregular rhythm. Because of its outstanding clinical feature, the *completely irregular pulse*, pulsus irregularis perpetuus,

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Heart Beat. Ed. 3, Lond., 1925, in which these early articles are listed with references to text comments.)

[1] Jolly, W. A., and Ritchie, W. T. Auricular flutter and fibrillation. *Heart*, Lond., 1910–11, ii, 177–221. (P. 478, ref. 429.)

[2] Ritchie, W. T. Auricular flutter. *Edinb. M. J.*, 1912, n.s., ix, 485–505. (P. 502, ref. 790.) Further observations on auricular flutter. *Quart. J. Med.*, Oxford, 1913–14, vii, 1–12. (P. 502, ref. 792.)

[3] McWilliam, J. A. Fibrillar contraction of the heart. *J. Physiol.*, Cambridge, 1887, viii, 296–310. (P. 492 #650.) (Figures in parentheses refer to the pages and reference numbers in Lewis, T. Mechanism and Graphic Registration of the Heart Beat. Ed. 3, Lond., 1925, in which these early articles are listed with references to text comments.)

pulsus arrhythmicus and delirium cordis, were but a few of the many, now obsolete, names applied to this condition.

The chief progress in the study of auricular fibrillation began with the polygraphic technique and clinical observations of James Mackenzie in 1902 when he published "The Study of the Pulse."

As an explanation of the mechanism of auricular fibrillation, Mackenzie offered the hypothesis that the cardiac stimulus arose in the auriculo-ventricular node; accordingly he termed the disturbance "nodal rhythm." This hypothesis was soon abandoned, however, even by Mackenzie.

Other attempts have been made to explain the mechanism of this peculiar disorder. The hypothesis holding sway prior to 1920 explained fibrillation on the assumption that multiple ectopic foci in the auricles gave rise to multiple stimuli which induced contractions of independent groups of the auricular musculature; of these stimuli, it was further supposed, only a certain number succeeded in inducing ventricular responses, resulting in a highly haphazard, irregular heart beat.

Mechanism of Auricular Flutter and Auricular Fibrillation Explained on the Theory of Circus Movements

In accordance with the most recent view on the subject, substantiated by abundant experimental evidence, the nature of the disturbance in the intrinsic cardiac mechanism in the case of auricular flutter and fibrillation is explained on the theory of the mechanism of "circus movements," as originally observed in the experiments of Mines[1] and Garrey.[2]

[1] Mines, G. R. On the summation of contractions. *J. Physiol.*, Lond, 1913, xlvi, 1–27; *Tr. Roy. Soc. Canada*, 1914, viii, 43.

[2] Garrey, W. E. The nature of fibrillary contraction of the heart; its relation to tissue mass and form. *Am. J. Physiol.*, Bost., 1914, xxxiii, 397–414.

Lewis and his collaborators, investigating the phenomenon of circus movements in relation to clinical flutter and fibrillation, have concluded that the fluttering or fibrillating auricles are activated by a self-perpetuating wave of excitation traveling in a closed, circuitous path at a very rapid rate, and that the path of such excitation wave is situated within the auricular muscle.[1]

Explanation of Diagrams Showing Circus Movements. Diagrams A, B, C, D in Figure 51 may serve to elucidate this mechanism.

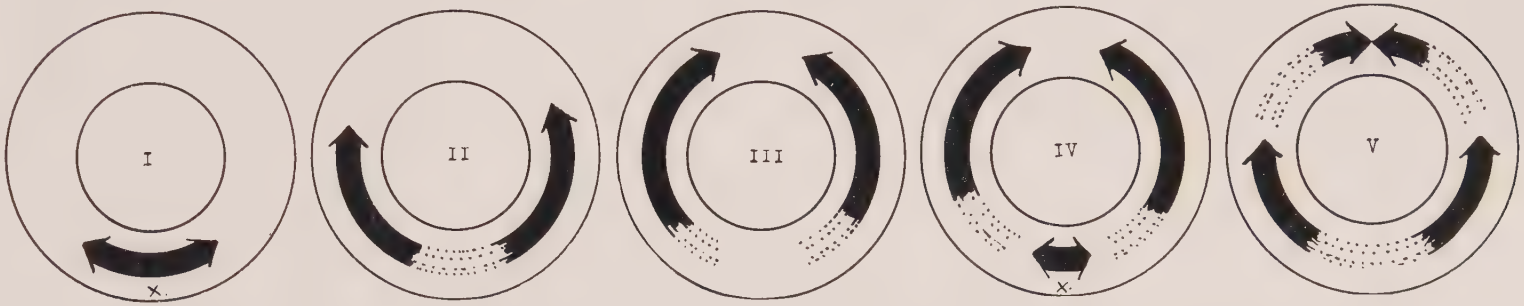
A-I represents a ring of muscle in which a stimulus is applied at X. If the muscle is functionally intact, the impulse spreads from point X in both directions with two advancing crests, as shown in A-II and A-III until their respective crests meet and extinguish each other, as shown in A-V.

In the wake of the impulse, the muscle remains refractory for a short time; this is indicated by the dotted shading in A-II. Depending upon its physiological integrity such muscle tends to recover (A-III). The time of recovery may vary, but once recovered, another stimulus may be applied to it at the original Point X (A-IV). Such second impulse naturally follows in the wake of the former (A-V). If the recovery of the muscle is prompt, that is, if its refractory phase is short, successive stimuli may be applied to it at a comparatively rapid rate.

Such rapid re-excitation of a ring of muscle may bring about a condition of partial refractoriness; in other words, some part, or fraction of it, may fail to recover in time to receive the advancing stimulus, so that a *temporary functional unidirectional block* may occur. This is shown at Y in B-I. Such a

[1] Lewis, T. The nature of flutter and fibrillation of the auricle. *Brit. M. J.*, 1921, i, 551–555.

FIG. 51.—Diagrammatic representation of the mechanism of Circus Movements. Figures in A, B, C and D represent circular masses of muscle; the heavy shaded areas within them denote the course of a contraction wave traversing circuitous muscular paths; the dotted shadings indicate unrecovered or refractory musculature in the wake of the contraction wave. The clear areas within the circle represent receptive (recovered or excitable) musculature.



A. In Circle I, a stimulus is applied at X, and a contraction wave is set up which travels in two directions, leaving the muscle refractory in its path as seen in II. In III, the refractoriness is shown to have recovered. In IV another stimulus is applied at the original point X and the second contraction wave is seen to follow in the wake of the first one. The crests of these finally meet and extinguish each other as shown in V.

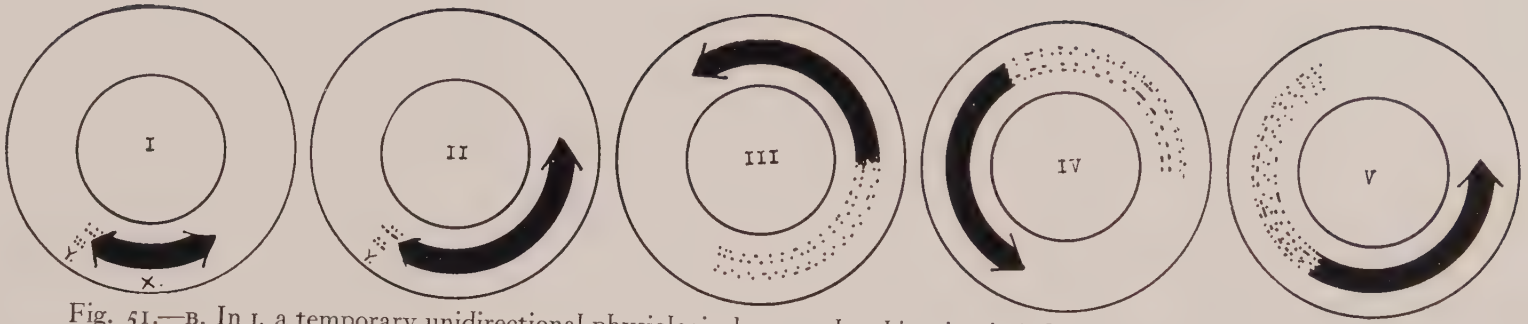


Fig. 51.—B. In I, a temporary unidirectional physiological block is indicated at point Y (dotted shading). A stimulus applied at X can travel, therefore, in one direction only, as shown in II and III. In IV, the contraction wave, having completed its circuit (Y having meanwhile recovered) reenters the point where it originally started, passes it (V) and continues as a self-perpetuating ring of excitation as long as there remains a gap of receptive (recovered) muscle in its path.

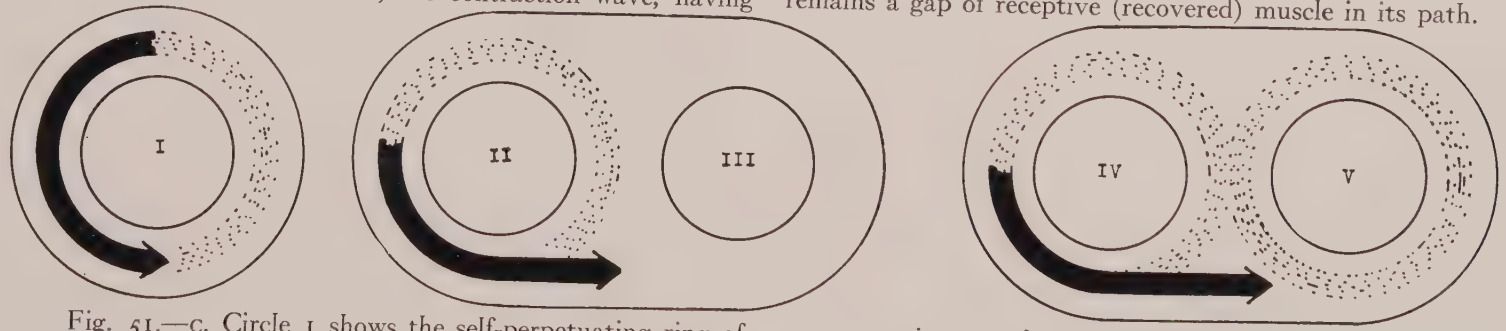


Fig. 51.—C. Circle I shows the self-perpetuating ring of excitation blocked by total refractoriness of the musculature. The circus movement is therefore terminated. II and III are adjacent rings of muscle, one of these having served as a path for a circus excitation wave and, having become successively more and more refractory (until there is no more receptive gap of muscle), tends to terminate the mechanism. However, in this case, the stimulus may invade the adjacent ring and may perpetuate itself in the adjacent path or in the long path encircling both rings. IV and V show both paths refractory, leading to a final termination of the circus movement.

stimulus can travel, therefore, in one direction only and therefore with a single advancing crest (B-II). Having no opposing wave in its path, it tends to travel onward in the circuit as indicated in B-III and B-IV. If by the time it has reached the

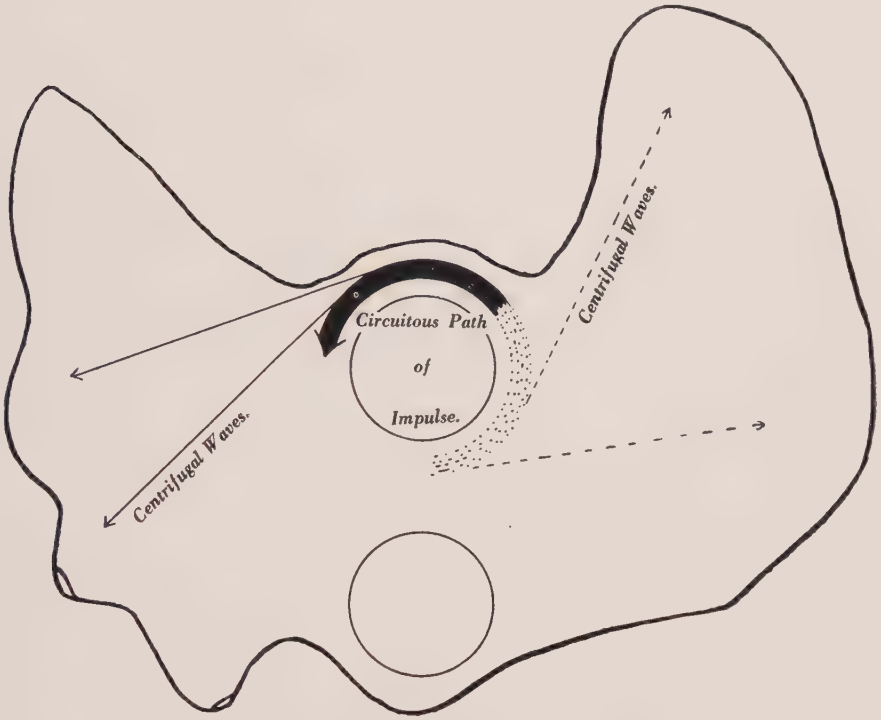


FIG. 51D.—A schematic diagram representing a mass of muscle within which there are two available circuitous paths suitable for the mechanism of circus movement. A wave of excitation is seen to travel in one of these paths (it may travel in either of the two or in both paths). In its course, the central ring of excitation, "mother ring" sends centrifugal impulses to the adjacent musculature activating it to contract with a tempo corresponding to the rate at which the circus movement traverses its path per minute.

point of the temporary block at Y, the partial refractoriness has recovered, the impulse invades this area (Y), passes it and *reenters the original point of stimulation at* X, namely the point

where it started. Thus it may continue in its closed circuitous path as a self-perpetuating ring of excitation for a varying length of time, as long as there is a gap of receptive (recovered) muscle between its crest and wake.

In experiments, such a mechanism was seen to continue for many hours. In the human heart, we have reason to believe that such a mechanism may go on for years with remarkable constancy.

Conditions Essential to Perpetuation of Circus Movement. The perpetuation of circus movement depends upon the maintenance of a gap of receptive muscle between the crest and the wake of the excitation wave. The maintenance of such a gap depends upon certain factors:

1. The length of the path.
2. The duration of the refractoriness (rate of recovery).
3. Rate of conduction (rate of propagation of impulse).

The path being generally constant for a given disorder, the actual determining factors that control the length of the gap are factors 2 and 3. If either the refractory period increases (delayed recovery in the wake of the impulse) or if the rate of propagation increases (impulse travels faster than muscle recovers), the tendency for the gap is to shorten and finally to disappear, thus bringing the mechanism to an end (Fig. 51 C-I).

But if there is an adjacent, similar path of muscle, as shown in C-II and C-III, the stimulus may invade it and perpetuate itself in a new path whose extent includes both the original path and the one adjacent to it; so that the stimulus now travels in a larger, closed path. Here it may continue for a time, because, even though the rate of conduction is increased or recovery delayed, nevertheless, in such longer path there may still remain an appreciable gap of receptive muscle

between the crest of the advancing wave and its wake. However, if the conditions that favor increasing refractoriness or an increasing rate of conduction persist, the gap will gradually shorten until eventually the crest of the excitation wave will find no responsive muscle even in this long path, and thus, here too, the mechanism will tend to terminate (C-IV and C-V).

If the circuitous path of muscle in which a self-perpetuating wave has been set up is a part of a larger mass of muscle (see Fig. 51D.), the "central ring" or "mother ring" of excitation sends centrifugal waves to the adjacent muscle mass, activating it to contract at a tempo which corresponds to the rate with which the circus movement completes itself per unit time. For example, if the central excitation wave traverses its closed path 300 times per minute, the adjacent musculature will contract 300 times per minute.

The arrangement of the musculature around the orifices of the great veins and around the orifices that separate the upper and lower cardiac chambers, in the mammalian and human hearts, is such as to offer a favorable path for the perpetuation of circus movements (Fig. 52).

Lewis, in an ingenious experiment, traced the path of the impulse in an experimentally induced flutter in a dog's auricle. He found that the circuitous path of the excitation wave approximately encircled the mouths of the superior and inferior venae cavae (Fig. 53).

Application of Theory of Circus Movement to Clinical Auricular Fibrillation and Flutter. It may be said in summary that there is abundant experimental proof to indicate that, in auricular fibrillation and auricular flutter, the intrinsic mechanism in the auricles is profoundly altered; that instead of the cardiac pacemaker setting the pace for the heart, there

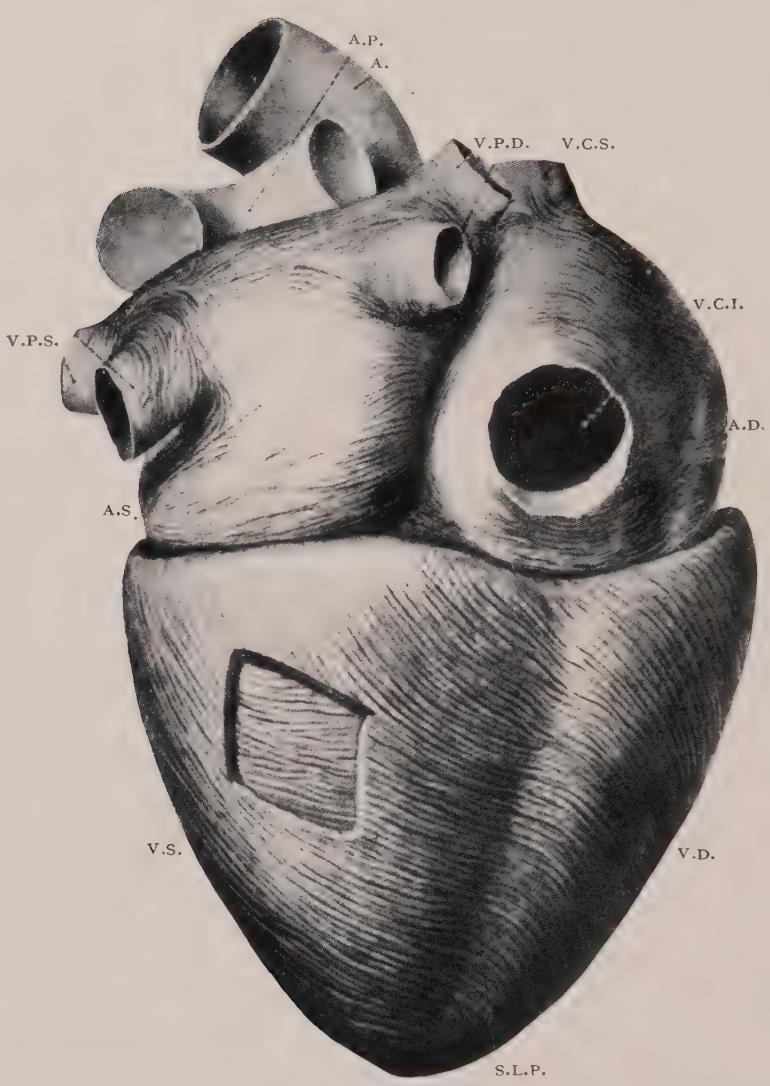


FIG. 52.—Posterior view of the heart, displaying the arrangement of the musculature around the orifices of the great veins. Such arrangement renders these structures particularly suitable as paths for "circus movements." A, aorta; A.D., atrium dextrum; A.P., arteria pulmonalis; A.S., atrium sinistrum; S.L.P., sulcus longitudinalis posterior; V.C.I., vena cava inferior; V.C.S., vena cava superior; V.D., ventriculus dexter; V.P.D., venae pulmonales dextrae; V.P.S., venae pulmonalis sinistrae; V.S., ventriculus sinister. (*From Sobotta.*)

is set up within the auricles a self-perpetuating ring of excitation which travels in a closed circuitous path at a very rapid rate and that it is the rate of this wave of excitation that determines the rate of the auricular responses.

While the mechanism of auricular flutter and fibrillation are fundamentally similar, they differ, nevertheless, in some essential features.

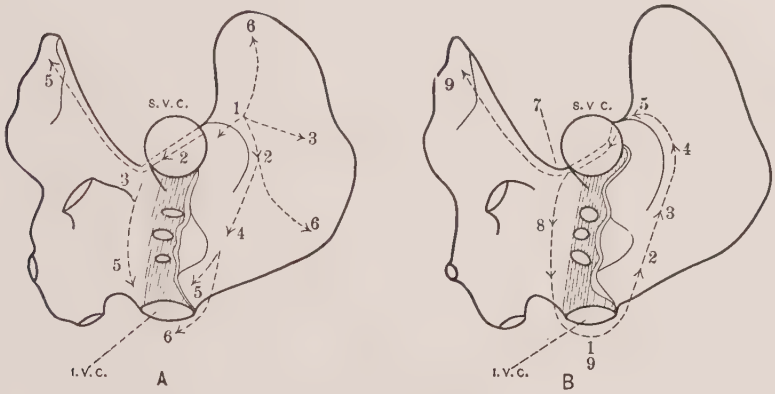


FIG. 53.—Diagram of posterior portion of auricle. A, order of excitation when impulse spreads normally from S-A node to right and left auricles. B, order of excitation in "circus movements" during flutter. (*After Lewis.*)

The path of the *circulating wave in flutter is longer and is constant.* It generally encircles the mouths of the superior and inferior venae cavae. *In fibrillation,* on the other hand, *the path is shorter and inconstant* (*shifting path*), generally encircling but one orifice.

As will be seen, these differences are potent factors in determining the different clinical manifestations of these two types of disorder.

Figure 54A is a diagram of the transparent heart in which the heavily shaded areas represent the specific system.

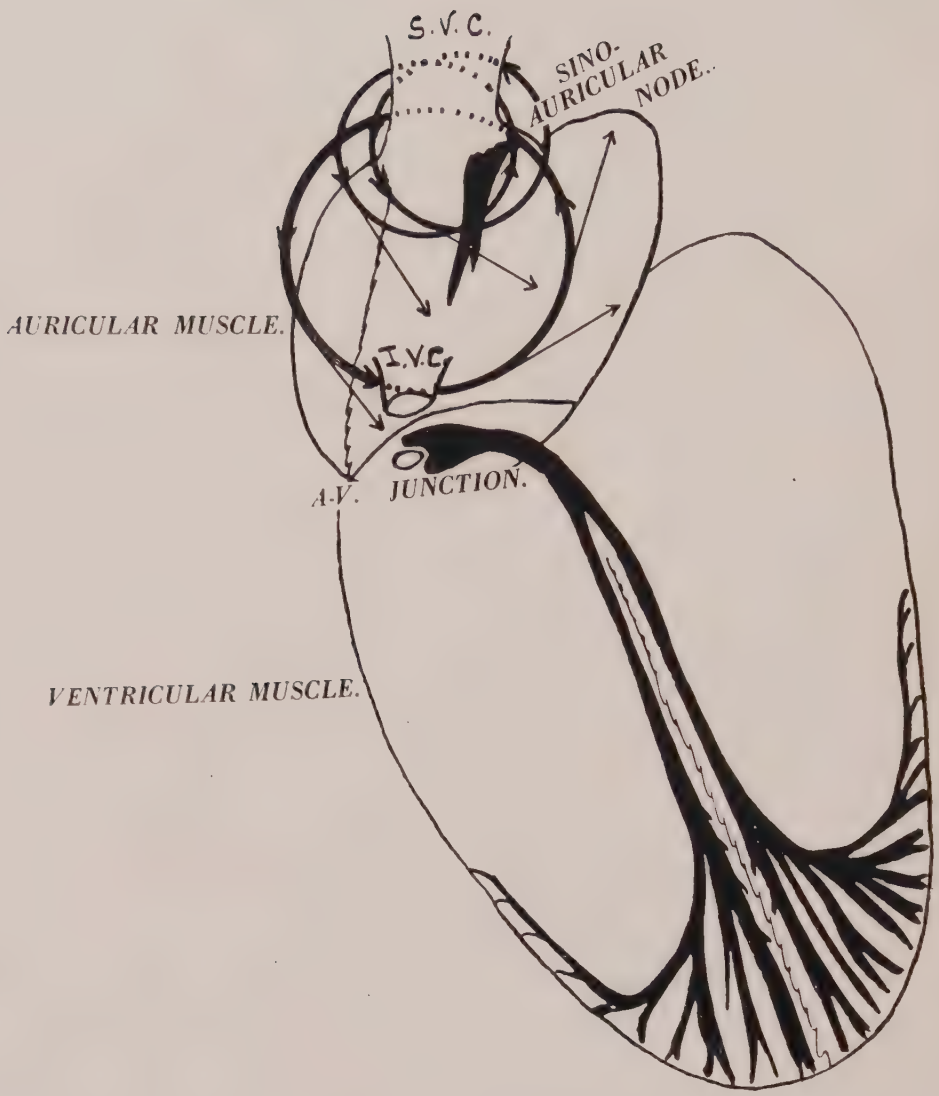


FIG. 54A.—Schematic diagram of a transparent heart in which the heavy shaded areas represent the specific system. The circles in the region of the auricles represent potential circuitous pathways for a self-perpetuating impulse. The large one encircling the mouths of the superior and inferior venae cavae indicates the long impulse path in flutter: the small ones grouped about the mouth of the superior vena cava indicate the short shifting pathways of the impulse in fibrillation.

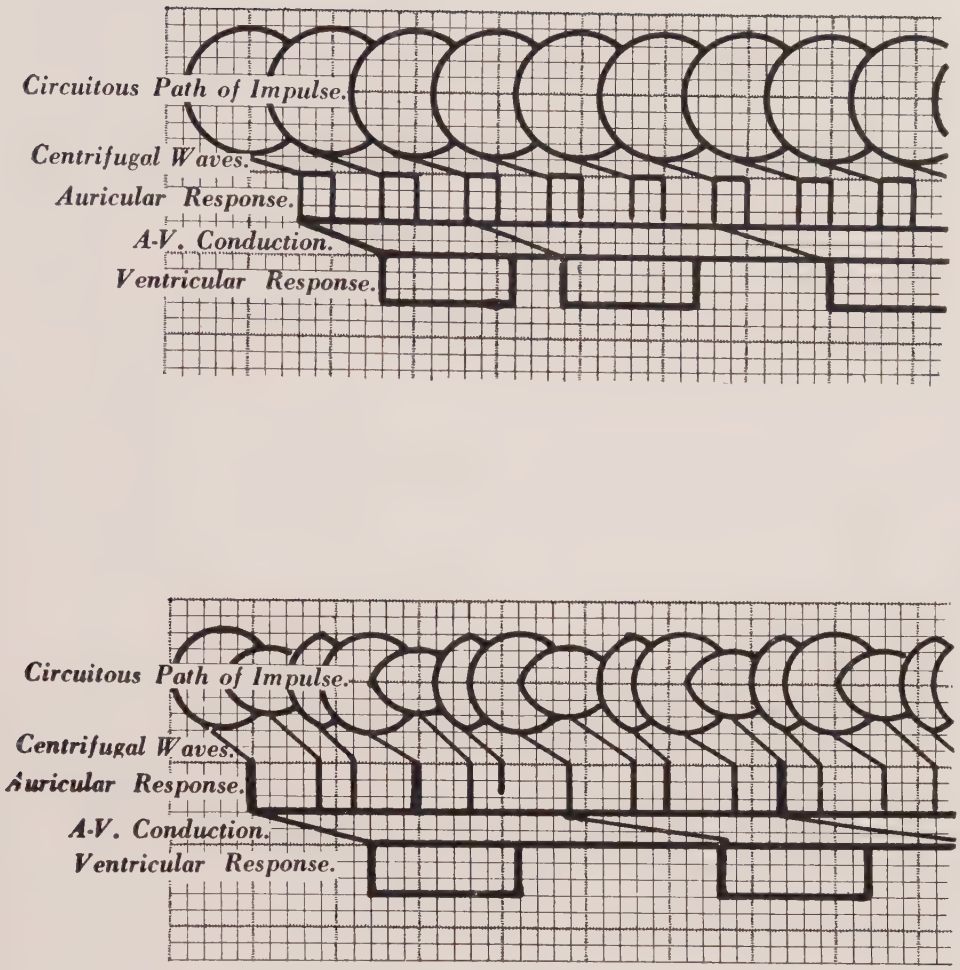


FIG. 54B.—Upper. Diagrammatic representation of the intrinsic cardiac mechanism in auricular flutter. (See text p. 168.)

Lower. Diagrammatic representation of the intrinsic cardiac mechanism in auricular fibrillation. (See text p. 168.)

In addition, it shows four circular paths, denoting diagrammatically four potential circuitous paths of a self-perpetuating impulse. The large one, encircling the mouths of the upper and lower venae cavae, indicates the impulse path in flutter. The three smaller ones grouped about the mouth of the superior vena cava indicate the paths of the stimulus in fibrillation.

In Figure 54B the upper and lower diagrams represent, diagrammatically, the mechanism of auricular flutter and auricular fibrillation on the basis of the theory of circus movement. The vertical lines in the background of the diagram represent time: heavy lines equal $\frac{1}{5}$ second and the fine lines equal $\frac{1}{25}$ second.

In the upper diagram (54B), the large uniform circles indicate the circuitous paths of a self-perpetuating excitation wave in which the stimulus *completes each circuit uniformly*, at a rate of $\frac{1}{5}$ of a second, or 300 times per minute (the approximate rate of circus movements in flutter in the human heart). The oblique lines extending from these circles represent the centrifugal waves that invade the adjacent auricular musculature. The small blocks represent the rhythmic dynamic auricular responses at a rate which corresponds to the number of circus movements completed per minute.

The A-V junction is shown to block some stimuli and to permit others to pass to the ventricles and thus to initiate ventricular responses. Depending upon the physiological integrity of the A-V junction, the *ventricles may follow rapidly or slowly, regularly or irregularly*.

The lower diagram is presented to depict similarly the mechanism of auricular fibrillation. The small, constantly varying circles and ovals represent the short and constantly shifting, but nevertheless, closed circuitous paths of the excita-

tion wave in which the stimulus traverses the circuits successively at the approximate rate of 450 per minute. Because of the short path, the rate is rapid; because of the shifting path, the individual circuits are *not completed at a uniform rate* and therefore the centrifugal waves, extending from them, activate the adjacent musculature at a very rapid and highly irregular rate. The response of the auricular musculature is represented as fractionate contractions, so that, as the diagram indicates, an effectual auricular contraction is practically never present (see auricular response in flutter for contrast).

The stimuli that reach the junctional tissues are very numerous and since they arrive at an absolutely irregular rate fundamentally they tend to induce an irregular ventricular response (for contrast see ventricular response in flutter). The A-V junctional tissues cannot transmit all these stimuli to the ventricles; furthermore, many of those transmitted reach the ventricles in their refractory stage and are therefore ineffective.

Depending upon its physiological integrity, the A-V junction may intercept a lesser or greater number of these impulses and consequently the ventricular responses may be rapid or slow, but *almost always irregular.*

Clinical Features of Auricular Flutter

Auricular flutter is generally regarded as an infrequent cardiac anomaly. This condition probably occurs more frequently, however, than is generally supposed: it is probably often overlooked. The reason for such oversight is twofold: in the first place, auricular flutter presents but few clinical signs; secondly, the condition generally appears with a perfectly regular ventricular rhythm and therefore escapes attention.

FIG. 55.—Diagrammatic representation of the mechanism and extracardiac manifestations of Auricular Flutter (with periods of regular ventricular response).

A, *Mechanism:* The excitation wave traverses a large circuitous path (surrounding the mouths of the upper and lower venae cavae) uniformly at a rate of 300 per minute, sending off centrifugal waves to the adjacent auricular musculature at the same rate. The auricles contract rhythmically at a rate of 300 per minute. The junctional tissues consistently block some of the impulses going to the ventricles, thus cutting down the ventricular responses. When every second impulse is consistently blocked, the ventricles beat regularly at a rate of 150 per minute (ventricular cycles 1 to 5 inclusive): when every second and third impulse is blocked, the ventricles beat regularly at a rate of 100 per minute (ventricular cycles, 6, 7 and 8): when every second, third and fourth impulse is consistently blocked so that only one out of every four can bridge the junctional tissue the ventricles beat at a rate of 75 per minute (cycles 8, 10 and 11).

B, *E. C. G.:* Rhythmic auricular P waves at a rate of 300 per minute: they are large and prominent and overshadow every other complex. The initial (Q, R, S) is generally of normal configuration, distorted only at its base by the prominent P waves. The terminal ventricular complex T is generally much distorted by the P waves so that as a rule it is barely visible.

C, *Heart Sounds:* The heart sounds are generally normal, modified only by the rate of the ventricular responses. If rapid, it may have a "tic-tac" quality.

D, *Jugular Pulse:* Visualization of the jugulars (more so in venous curves) often reveals the venous pulse to consist of a series of fine, regular and rapid undulations interrupted only by the ventricular contractions. In moderately slow hearts, during the long diastoles, this phenomenon is generally prominent and is a good diagnostic guide.

E, *Carotid Pulse:* The arterial pulse generally follows the ventricular rate. When rapid, the pulse may exhibit alternations.

AURICULAR FLUTTER

(*With 2:1, 3:1 and 4:1 block*)

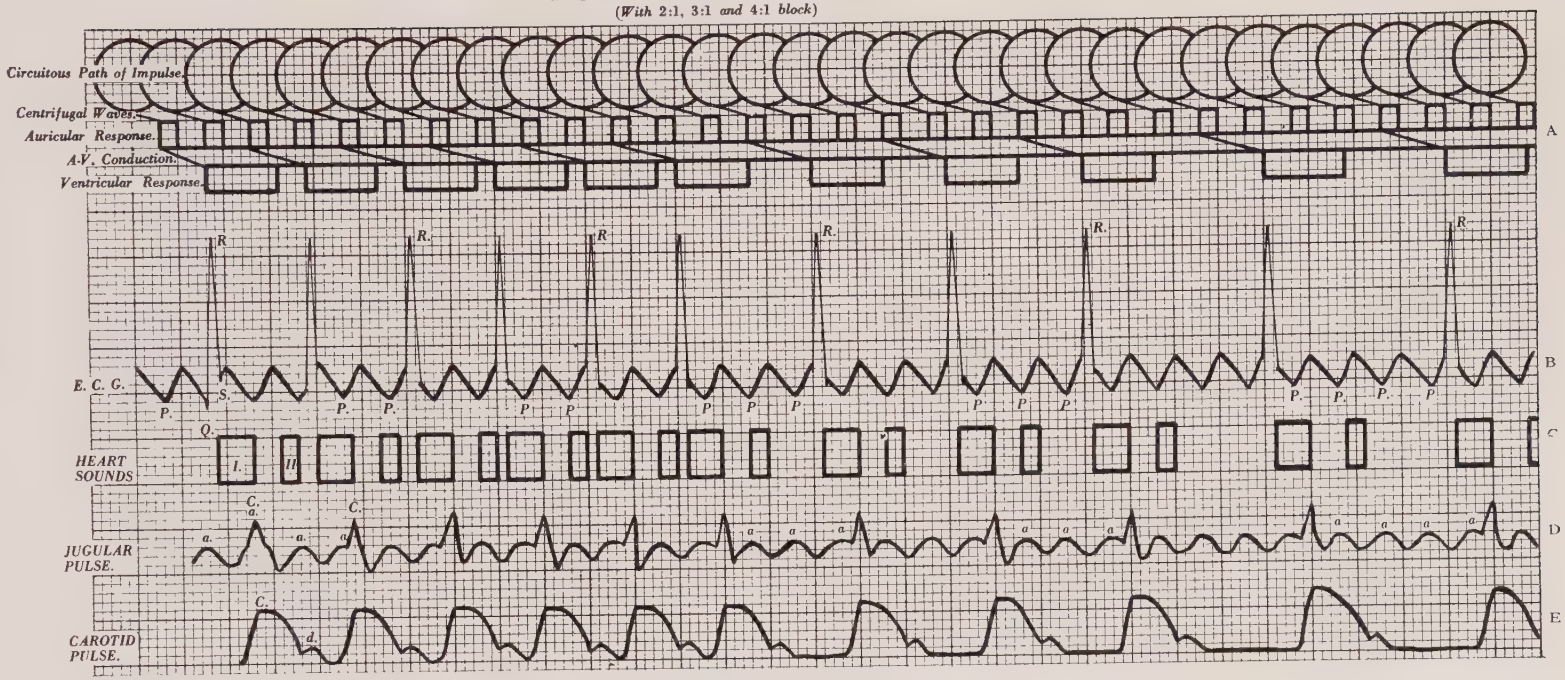
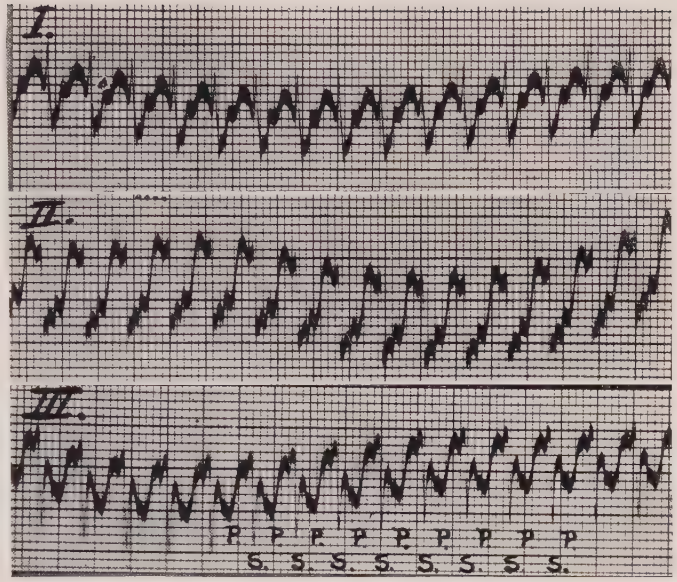


FIG. 55.

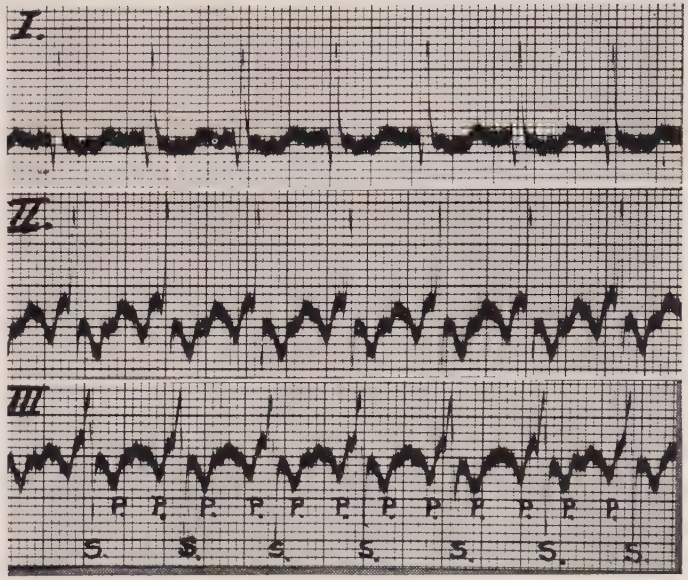
However, if one understands its mechanism and is prepared to recognize its clinical manifestations, and if one realizes the fact that it is not as infrequent as is generally supposed, a diligent search for it not infrequently rewards one with the pleasure of having detected it, or at least of having suspected it sufficiently to institute graphic measures. Auricular flutter not infrequently occurs in sclerotic hearts and the disturbance may go on for years undetected. It may occur as a transitory condition in chronic rheumatic valvular defects prior to the onset of fibrillation; also as a transitory condition in hearts that emerge from fibrillation, as when treated with quinidine.

Clinically, auricular flutter may manifest itself as a mere tachycardia with a regular rate of approximately 150 (130 to 160). This rate corresponds to half the rate of the number of circus movements in the fluttering auricles. At times, the ventricular rate may be slower and still regular. The rate may be 100 or even 75 per minute in which case the rate approximately corresponds to one-third or one-fourth of the rate of circus movements in flutter (Fig. 55). Change of posture or slight effort elicits no change in the rate.

If a case of auricular flutter with a moderate or slow ventricular rate is exercised, the acceleration does not take place gradually but *changes suddenly* to an approximate rate of 150, the usual ventricular rate in flutter. This is due to the fact that instead of the ventricles responding as they do in the slower types to every third or fourth supraventricular stimulus, they respond instead to every second. A rate of 150 is usually the highest ventricular rate attained in flutter. It ordinarily denotes the maximum efficiency on the part of the junctional tissues. There are rare cases on record however, in which, following exercise, the heart rate suddenly changed to 300 per



A



B

FIG. 56A and B. (A) Auricular flutter with a 1:1 auriculo-ventricular ratio. Each auricular impulse is followed by a ventricular response. The duration of a cardiac cycle equals 0.24 seconds. The fluttering auricles beat at a rate of 283.33 per minute, giving rise to an equal number of ventricular contractions. The paroxysm in this case was associated with marked signs of circulatory insufficiency.

(B) Auricular flutter with a 2:1 auriculo-ventricular ratio which the same patient had before the onset of the attack of tachycardia shown in A.

FIG. 57.—Diagrammatic representation of the mechanism and extracardiac manifestations of Auricular Flutter (with an irregular ventricular response).

A, *Mechanism:* The excitation wave traverses a large circuitous path (surrounding the mouths of the upper and lower Venae Cavae) uniformly at a rate of 300 per minute, sending off centrifugal Waves to the adjacent auricular musculature at the same rate. The auricles contract rhythmically at a rate of 300 per minute. The junctional tissues block some of the impulses going toward the ventricles thus cutting the ventricular rate far below that of the auricles. The conductivity of the junctional tissues being unstable the degree of block varies from beat to beat, so that while at times every second impulse may bridge the A-V junction at other times only every third, fourth or fifth may succeed in inducing a ventricular response. This inconsistency on the part of the junctional tissues renders the ventricular beat highly irregular (cycles 1 to 7 incl.). At times, if conduction improves temporarily, a short period of "dominant rhythm" may be detected at a regular, rapid rate (cycles 8, 9 and 10).

B, *E. C. G.:* Prominent, uniformly spaced P waves, appearing at rate of 300 per minute overshadow every other complex. The Q, R, S complexes are irregularly spaced in the first 7 cycles (irregular ventricular response). They are closely and uniformly spaced in the last 3 cycles (short period of "dominant rhythm" at a rate of 150 per minute).

C, *Heart Sounds:* The heart sounds generally follow the rhythm of the ventricles. Their character may become altered when the heart rate is accelerated.

D, *Jugular Pulse:* The venous pulse shows a series of fine, rapid and regular indulations, most marked during the long diastoles.

E, *Carotid Pulse:* The arterial pulse is irregular in rhythm and size. A pulse deficit is but rarely present.

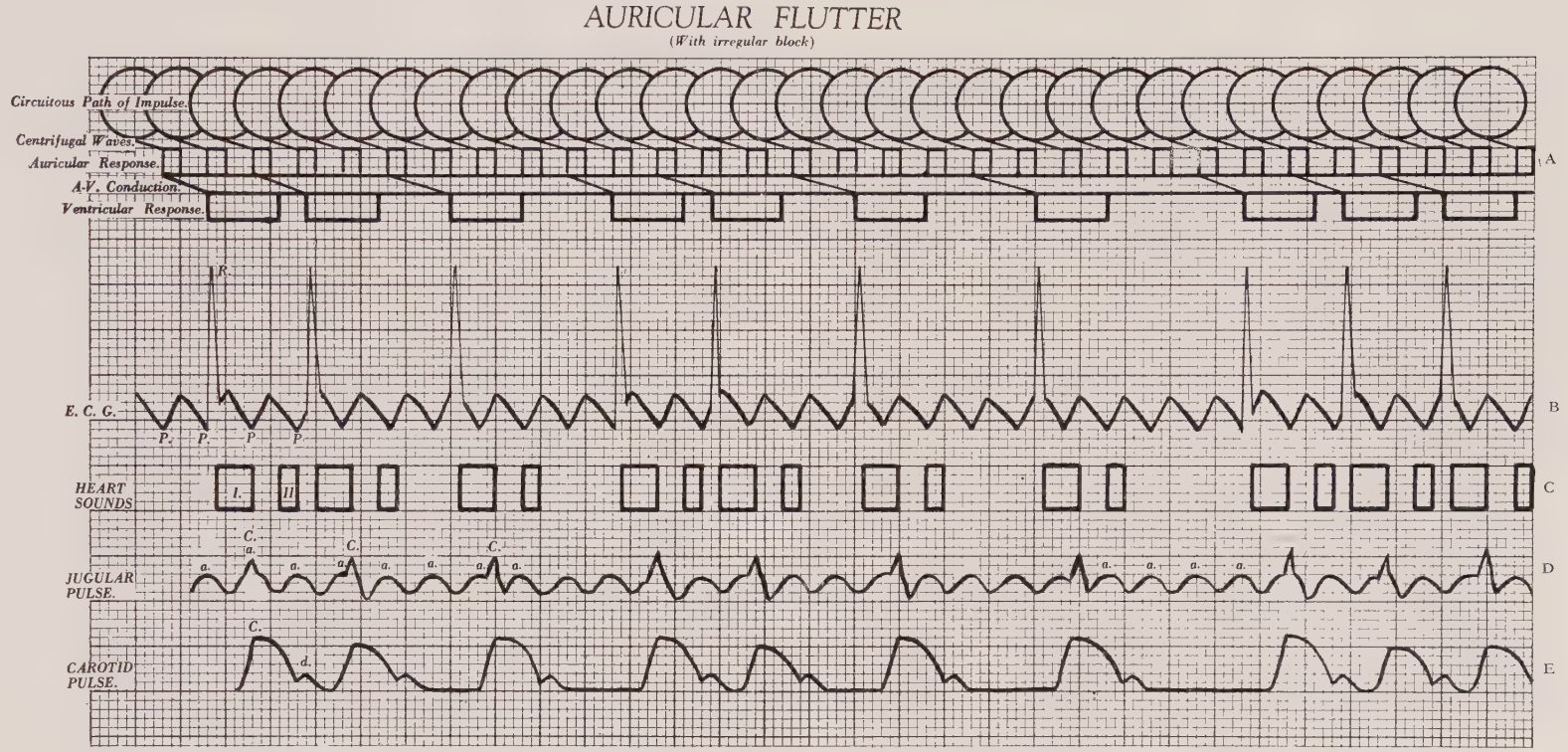

AURICULAR FLUTTER
(With irregular block)
Circuitous Path of Impulse.
Centrifugal Waves.
Auricular Response.
A-V. Conduction.
Ventricular Response.
A
R.
E. C. G.
P.
B
HEART SOUNDS
I.
II
C
C.
a.
JUGULAR PULSE.
D
CAROTID PULSE.
d.
E

FIG. 57.

minute for short periods. Such a rate corresponds to the full rate of circus movements in the fluttering auricles and presupposes perfect A-V conduction. It is a grave condition and associated with marked clinical signs of heart failure (Fig. 56).

Auricular flutter at times manifests itself, clinically, as a complete irregularity of the ventricular beats (Fig. 57). The clinical recognition of such cases is comparatively easy. Such hearts, when exercised, *tend to become regular and rapid* and the resulting ventricular rate is approximately 150 per minute (see the effect of exercise on fibrillation). On relaxation, following exercise, the heart generally becomes irregular again.

The heart sounds in flutter present no notable variations from the normal. They generally follow the rate and rhythm of the ventricles.

The arterial pulse also generally follows the ventricular rate and rhythm. At times, it may exhibit alternation. If the ventricles are irregular, the arterial pulse may show the irregularity not alone in rhythm but also in size, resembling in this respect the pulse in auricular fibrillation (see pulse in auricular fibrillation). A "pulse deficit," however, is not the rule but rather the exception in auricular flutter.

Visualization of the jugular vein in the neck often serves to suggest the presence of auricular flutter. If the ventricular rate is comparatively slow, or particularly when the ventricular rate is irregular, with numerous long cycles, one may, during the long diastoles, detect fine, rapid undulations in the jugular vein. These are due to the transmission of the small, rapidly recurring intra-auricular pressure changes into the veins near the heart. Graphic records show that these small venous waves recur with a remarkable uniformity and regularity (jugular pulse in Fig. 57, cycle 7, and Fig. 58).

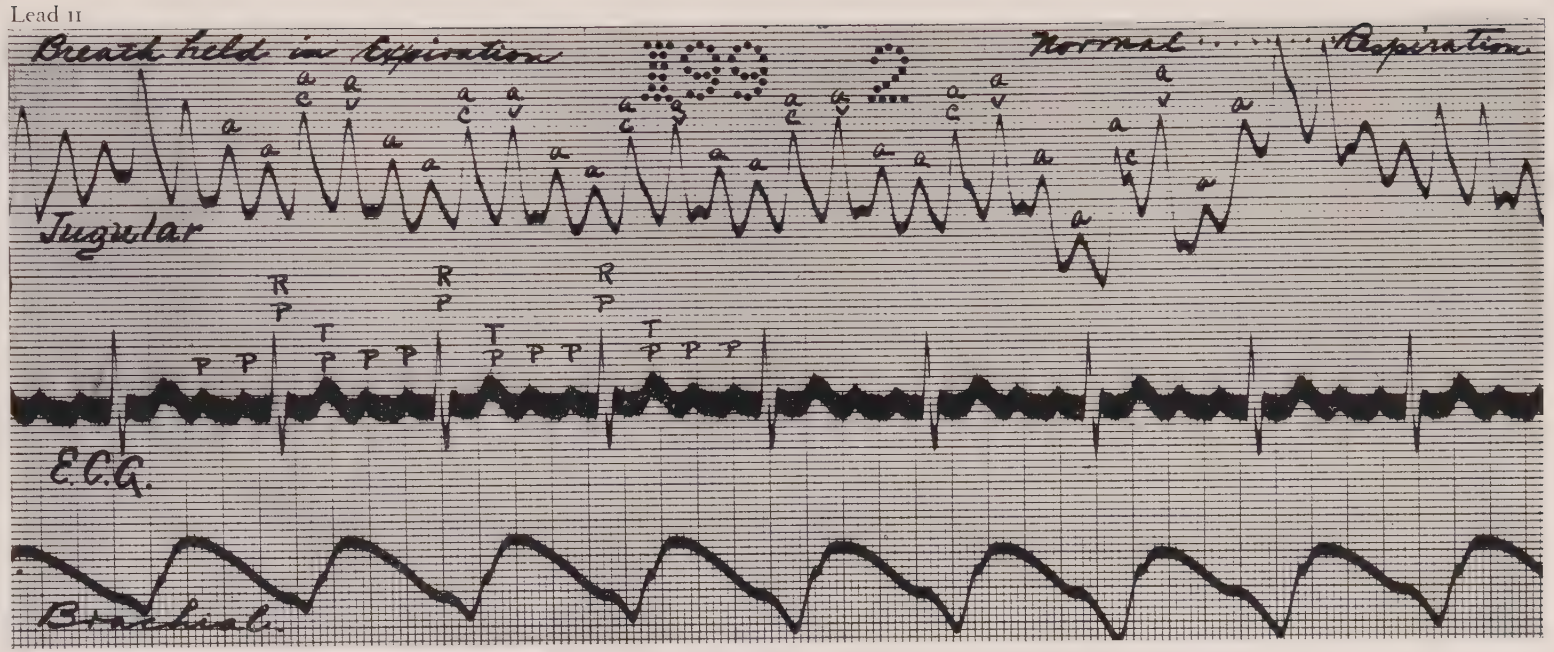


FIG. 58.—Jugular pulse in auricular flutter. Prominent rhythmic A waves in the jugular pulse tracing correspond to the rapidly recurring, equally spaced auricular P waves in the electrocardiogram. The brachial pulse beats correspond to the ventricular complexes. The three tracings were recorded synchronously.

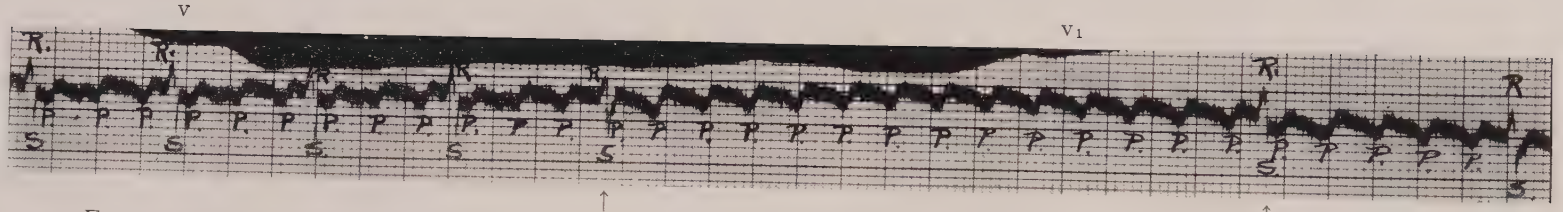


FIG. 59.—Electrocardiographic tracing of a case of auricular flutter in which vague pressure was applied at V, and maintained till V_1. Between vertical arrows (S–S) there is an unbroken series of P waves, indicating that while the vagus pressure had induced a temporary ventricular standstill, the flutter mechanism in the auricles was undisturbed.

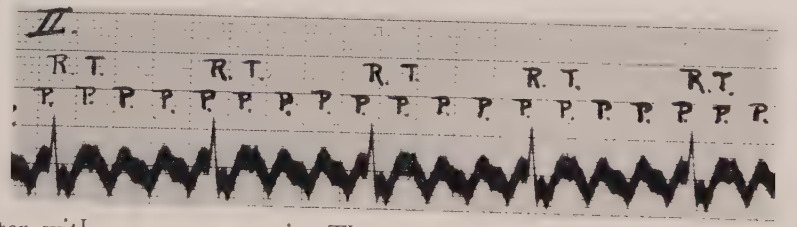


FIG. 60.—Auricular flutter with a 4:1 A-V ratio. The ventricular rate is regular. Clinically, such a heart may *appear* perfectly normal. The electrocardiogram *shows*, however, that the underlying mechanism is that of an auricular flutter. The prominent and uniformly spaced auricular P waves recur at a rate of about 300 per minute.

Figure 58 is a synchronous polygraphic and electrocardiographic tracing of a case of clinical auricular flutter with a 4:1 auriculo-ventricular ratio. Prominent rhythmic A waves in the jugular pulse tracing are seen to correspond to the rapid, rhythmic P waves, in the accompanying electrocardiogram.

Vagus stimulation, by firm pressure over the carotid artery in the neck, often produces temporary, profound slowing of the ventricles. Visualization of the jugulars, during such slowing, may show these fine, rapid undulations over a longer period. Vagus pressure does not affect the rate of flutter in the auricles. It merely blocks the junctional tissues and thus induces the marked ventricular slowing (Fig. 59).

The electrocardiographic curve in case of flutter is unmistakable. It shows uniformly spaced, distinct and prominent P waves overshadowing all other complexes. For their uniformity in spacing and constancy of form, even over long periods of time, these waves are truly remarkable (Fig. 60). They show best in leads II and III. In lead I, they are often obscured.

Clinical Features of Auricular Fibrillation

Auricular fibrillation in contrast to auricular flutter is a very frequently encountered clinical disturbance of the cardiac rhythm. It is frequently associated with sclerotic changes in the heart muscle, chronic rheumatic valvular defects, such as mitral and tricuspid stenoses and the myocardial changes in goiter. It may occur in short paroxysms and may therefore at times escape detection.

The most outstanding clinical feature of auricular fibrillation is the *perpetual irregularity of the heart beat* (Fig. 61).

The heart sounds generally lack all semblance of rhythmicity. Some sounds are loud and sharp, while others are weak.

FIG. 61.—Diagrammatic representation of the mechanism and extracardiac manifestations of Auricular Fibrillation (rapid ventricular rate).

A, *Mechanism:* A self-perpetuating wave of excitation repeatedly traverses a short circuitous path with a constantly changing outline (shifting path). Because of the shortness of the path, the circuits are traversed frequently (450 per minute). Because of the changing outline of the path, the time at which the circuits are completed is *not uniform.* The centrifugal waves issuing from the "mother ring" stimulates the adjacent musculature at a very rapid and highly irregular rate. The auricular musculature responds with faint fractionate contractions at a correspondingly rapid and irregular rate. Many of the stimuli are blocked at the A-V junction: those that come through induce ventricular responses at a somewhat rapid rate and an *absolutely irregular* rhythm.

B, *E. C. G.:* Ventricular complexes are irregularly spaced. There is no evidence of a P wave anywhere; instead there are seen, especially during diastoles, many fine, irregularly spaced wavelets, the "f" waves.

C, *Heart Sounds:* Total irregularity of the heart sounds, both in rhythm and intensity. When the heart is rapid, the second heart sounds are often much altered, some are short and sharp, others feeble and still others barely heard or entirely absent.

D, *Jugular Pulse:* The venous pulse lacks the multiple components (A, C, V) of the normal. Instead, it has a ventricular type of configuration.

E, *Carotid Pulse:* The arterial pulse is totally irregular both in rhythm and in size; large beats and small beats intermingle haphazardly. Many of the ventricular beats fail to reach the peripheral arteries and therefore the pulse beat falls below the rate of the ventricular beats. The difference between the ventricular rate and the arterial pulse rate constitutes the "pulse deficit."

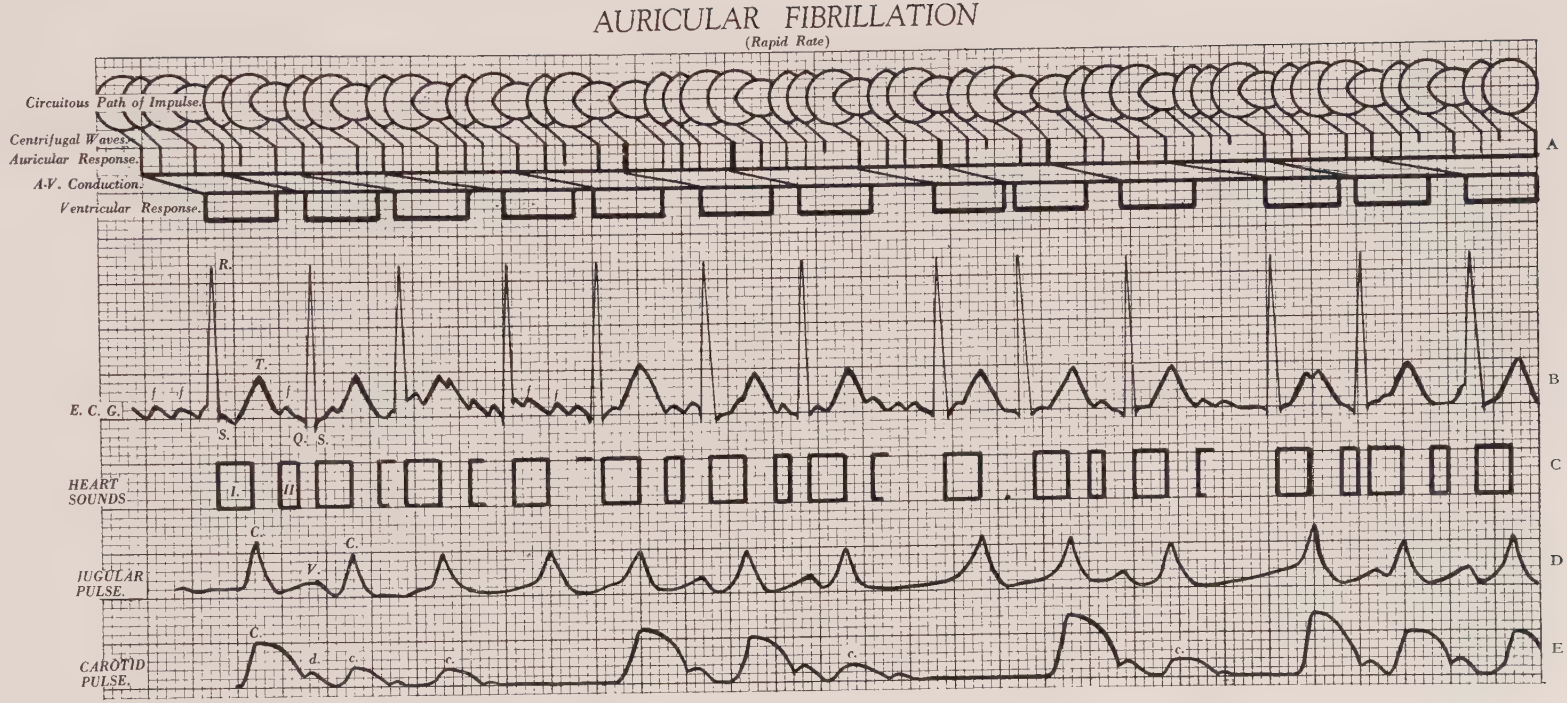

AURICULAR FIBRILLATION
(Rapid Rate)
Circuitous Path of Impulse.
Centrifugal Waves.
Auricular Response.
A-V. Conduction.
Ventricular Response.
A
R.
T.
E. C. G.
P.
Q.
S.
B
HEART SOUNDS
I.
II.
C
C.
V.
JUGULAR PULSE.
D
CAROTID PULSE.
d.
c.
E

FIG. 61.

FIG. 62.—Diagrammatic representation of the mechanism and extracardiac manifestations of Auricular Fibrillation (with a slow ventricular rate).

A, *Mechanism:* The excitation wave repeatedly traverses a small, closed circuitous path of a constantly changing outline. Because of the lack of uniformity in completing its successive circuits, the "mother ring" sends its centrifugal stimuli to the adjacent auricular musculature at a rapid and highly irregular rate (450 per minute). The auricular musculature responds with rapid, feeble, fractionate contractions. The conductivity of the junctional tissues is depressed (may be caused by digitalis) and therefore the resulting ventricular rate is slow but nevertheless totally irregular.

B, *E. C. G.:* Irregularly spaced ventricular complexes of normal configuration. There is no evidence of P waves anywhere, instead, there are seen, during the long diastoles, many fine irregular wavelets—the "f" waves. Because of the slow rate, the cardiac irregularity is not always apparent (cycles 4, 5, 6, and 7): careful measurements show, however, that the irregularity is absolute.

C, *Heart Sounds:* Irregularity of rhythm is readily noted; the character of the sounds is not much altered when the heart is slow.

D, *Jugular Pulse:* The venous pulse lacks the multiple components of the normal. It has a ventricular type of configuration.

E, *Carotid Pulse:* The rhythm of the arterial pulse is irregular. The size of the pulse beat varies but slightly. In auricular fibrillation with a slow ventricular response, there is generally no "pulse deficit."

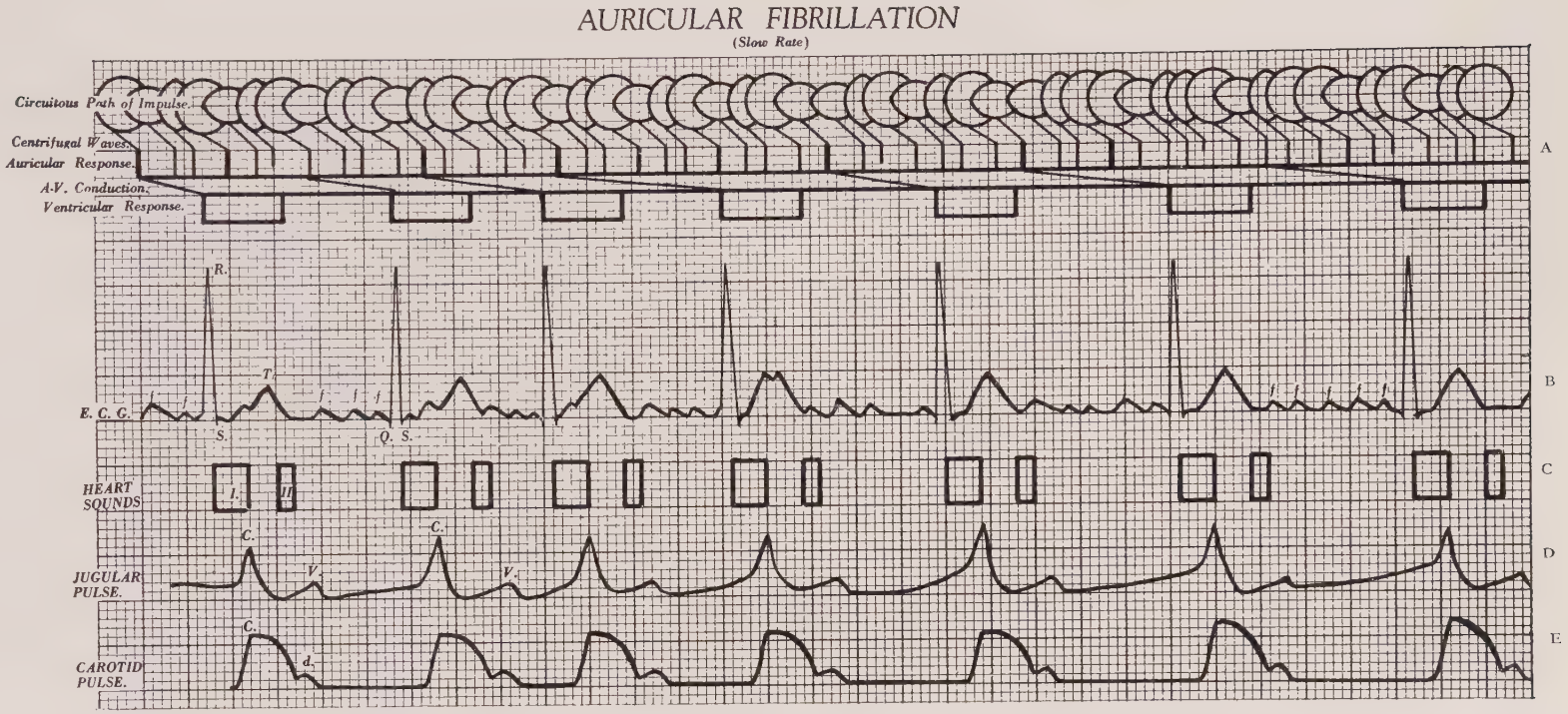

AURICULAR FIBRILLATION
(Slow Rate)
Circuitous Path of Impulse.
Centrifugal Waves.
Auricular Response.
A-V. Conduction.
Ventricular Response.
A
R.
T.
E. C. G.
S.
Q. S.
B
HEART SOUNDS
I.
II.
C
C.
V.
JUGULAR PULSE.
D
C.
d.
CAROTID PULSE.
E

FIG. 62.

The first sound may at times be followed by a sharp second sound; at other times by a very feeble second sound; still at other times, there may be no second sounds heard. This latter condition is especially true when the heart rate is rapid, in which case the heart sounds are very haphazardly spaced and tumultuous. If mitral stenosis is associated, the presystolic element of the diastolic rumble is generally absent (the auricles do not contract).

The arterial pulse presents characteristic features similar to those of the heart sounds. It is completely irregular both in rhythm and in size. Palpating such a pulse, one is readily impressed, not only with its absolute irregularity, but also with the frequent change in the type of the pulse beat. Large and small pulse beats intermingle haphazardly. The number of pulse beats do not, as a rule, correspond to the number of heart beats. Many do not reach the peripheral arteries and are, therefore, not felt at the wrist. A difference between the number of apex beats and of arterial pulse beats (the *pulse deficit*) is a common clinical manifestation of auricular fibrillation.

If the heart rate is slow, so that during the longer diastoles a substantial ventricular filling can take place, each ventricular contraction may induce a pulse beat at the wrist. (Fig. 62.) In auricular fibrillation with a slow ventricular response, therefore there is generally no pulse deficit, since there are no ineffectual heart beats. If mitral stenosis is associated with fibrillation, there is a tendency to a pulse deficit, even though the heart be moderately slowed, because in the presence of a stenosed A-V valve and a "paralyzed" auricle, diastolic ventricular filling is at best imperfect. Even temporary acceleration may lead to a marked pulse deficit in such hearts. On the other hand, if a case of auricular fibrillation is associated with

unimpaired mitral valves, the pulse deficit is generally at a minimum even though the heart is moderately rapid. This is particularly true in case of auricular fibrillation in chronic goiter hearts (Fig. 63).

In auricular fibrillation the pulse deficit is a good clinical guide in estimating the efficiency of the ventricular contractions. It is the aim of the clinician, in endeavoring to control the ventricular rate in this disorder, to reduce the pulse deficit to a minimum.

Visualization of the jugular pulse offers no aid in diagnosis. Polygraphic records do show, however, a definite alteration in the venous pulse. Since the auricles do not contract and consequently do not produce volume changes, the venous pulse lacks the A component of the normal A.C.V. curve. It has a ventricular type of configuration (jugular pulse in Figs. 61 and 62).

The electrocardiogram shows a complete irregularity in the spacing of the ventricular complexes. There is no evidence of a definite P wave anywhere. Instead, there is seen, especially during the diastoles, many fine, irregular wavelets following each other in rapid succession; the "f" waves.

Auricular fibrillation, with a slow ventricular rate, may give the impression of short periods of regularity (Fig. 62). In such a case, exercise may be used as a simple diagnostic test. Exercise increases the ventricular rate and *tends to enhance the characteristic irregularity* (see effect of exercise on auricular flutter with irregular ventricular response).

Conspicuous slowing of the ventricular rate in auricular fibrillation may be induced by digitalis administration. In fact, it is in auricular fibrillation that the most pronounced clinical effect of this drug is seen. Firm pressure over the carotid

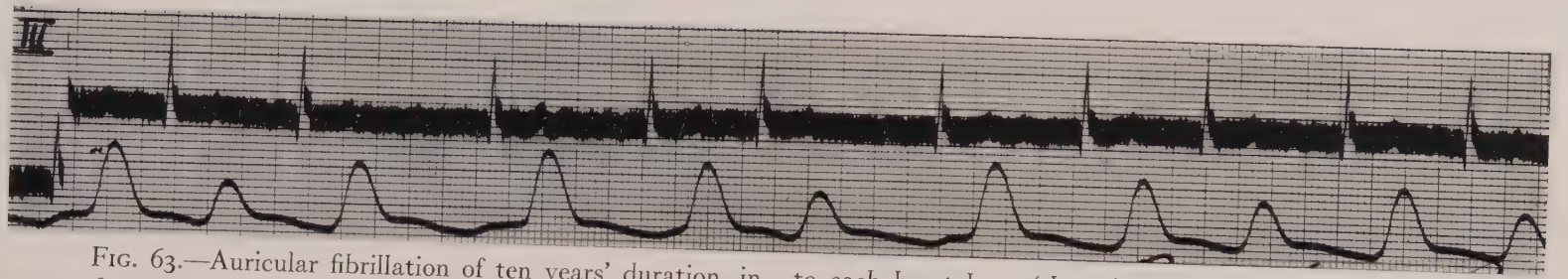


FIG. 63.—Auricular fibrillation of ten years' duration, in a chronic goiter heart, without valvular defect. With moderate precaution on the part of the patient, an efficient circulation is maintained, as shown by the perfect pulse response to each heart beat (there is no pulse deficit). This patient requires digitalis very rarely; in fact, he has not had any for three successive years.

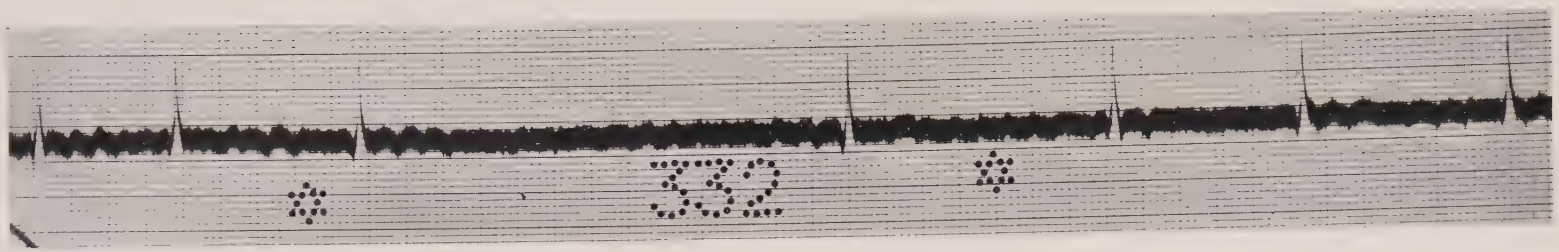


FIG. 64.—Auricular fibrillation in which right vagus pressure was applied (* to *). The ventricles are profoundly slowed. The fibrillation mechanism in the auricles is undisturbed.

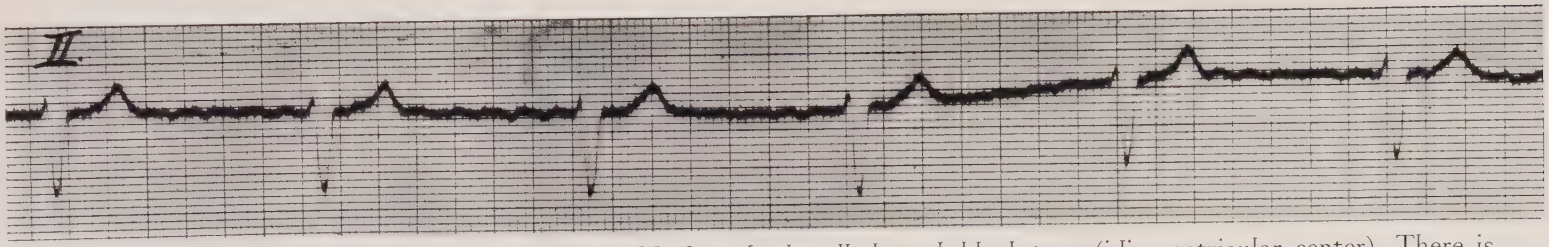


FIG. 65.—Complete auriculo-ventricular heart block and auricular fibrillation Ventricular rhythm is regular and the rate is 43 per minute. The ventricular complexes are the bundle-branch block type (idio-ventricular center). There is no evidence of a P wave anywhere; instead there are seen fine, rapid oscillations during diastole: the "f" waves.

sheath in the neck may also induce temporary slowing of the ventricles in auricular fibrillation (Fig. 64).

These effects are probably due to stimulation of the vagus which depresses the junctional tissues and impairs their conductivity. Atropine tends to eliminate the ventricular slowing in most of these instances, suggesting that the effect was primarily vagotropic in origin.

If there is a complete auriculo-ventricular heart block, associated with auricular fibrillation, the ventricular rate is very slow and in general regular. In such a case, the slow, regular ventricular rhythm is due to the fact that the fibrillating auricles do not influence the ventricles at all. There is an anatomical or physiological severance between the upper and lower cardiac chambers. The ventricles, therefore, respond to their own pacemaker: they manifest an "idio-ventricular rhythm" (Fig. 65).

CLINICAL FEATURES OF AURICULAR FLUTTER AND FIBRILLATION

	Hemodynamics			SPECIAL FEATURES
	Heart Sounds	Jugular Pulse	Arterial Pulse	
Auricular Flutter	Regular and rapid. Occasionally irregular.	Fine rapid oscillations during diastole.	Regular and rapid. Occasionally irregular.	Heart may be regular for long periods. When irregular, short periods of a "dominant rhythm," 140 to 150, may be detected. Exercise tends to *induce a regularity of rhythm.* Associated with atherosclerotic heart disease; long standing valvular defects.
Auricular Fibrillation	Complete irregularity of rhythm and intensity.	Ventricular form.	Complete irregularity of rhythm and size.	"Pulse deficit" may be present and is increased with increase in heart rate. Exercise tends to *enbance the irregularity.* Commonly associated with long-standing mitral or tricuspid stenosis; goiter; atherosclerosis.

Chapter XI

Combined Arrhythmias

IRREGULAR heart action of one kind may at times be accompanied by an irregularity of another class. The resulting combined arrhythmia is often difficult to recognize at the bedside, partly because of its increased complexity and partly because it does not conform to any given group of clinical criteria. On the other hand, the superimposition of one disorder upon another may render a previously irregular heart action perfectly regular and thus remove even a suspicion of the true nature of the original disorder.

Graphic measures usually reveal the nature of the components of the associated disorders. There are instances, however, in which even with the aid of instruments of precision a single examination is insufficient, and judgment has to be reserved until repeated examinations have been made, and the various phases of the disorder in question have been observed on successive days.

Auricular Fibrillation and Ventricular Extrasystoles

The association of ventricular extrasystoles with auricular fibrillation is the most common of all the combined arrhythmias (Fig. 66). It generally occurs in the course of excessive digitalis administration, given to control the rapid and irregular ventricular rate in auricular fibrillations. The extrasystoles usually appear when the ventricular rate is slowed and its irregularity lessened. The complicating extrasystoles superimposed upon the slow rate and apparently regular ventricular rhythm tend to render such a heart beat again irregular and if the combined disorder is not recognized and the clinician is guided

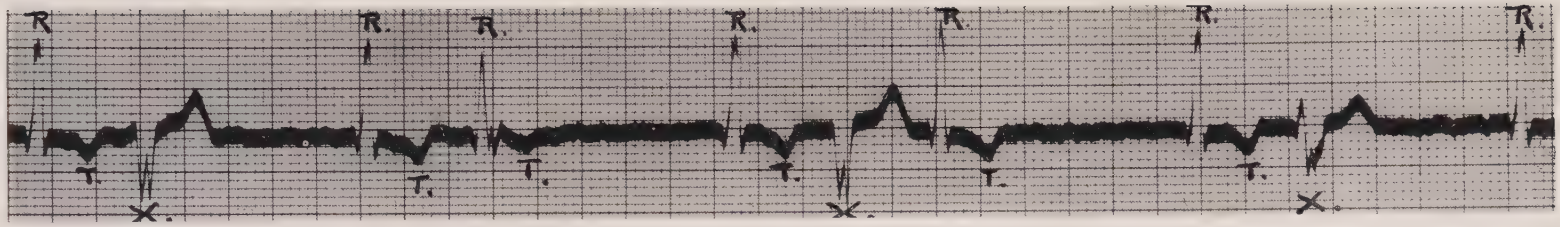


FIG. 66.—Auricular fibrillation and ventricular extrasystoles. The irregular spacing of the ventricular complexes and the absence of auricular P waves indicate that the fundamental irregularity is due to auricular fibrillation. In addition there are seen numerous bizarre ventricular complexes, ventricular extrasystoles (X).

only by the irregularity, an excessive amount of digitalis may be given. This combined arrhythmia may also appear in the course of quinidine therapy, employed with a view of stopping the mechanism of fibrillation of the auricles. In either case the appearance of the extrasystoles is a sign of excessive myocardial irritability and may be taken as a warning of impending drug intoxication.

Auricular Flutter and Ventricular Extrasystoles

In contrast to auricular fibrillation, auricular flutter is but rarely complicated by ventricular extrasystoles. The reason for the rarity of this coincidence is not entirely clear. It may be due to the fact that digitalis is not given as frequently and as freely in auricular flutter as in auricular fibrillation. On the other hand, it is more likely that, since flutter tends to be converted to fibrillation under the influence of digitalis, the stage of excessive myocardial irritability as evidenced by extrasystoles, is not reached during the stage of flutter but occurs, if at all, during the later stages of digitalization, when the transition to fibrillation has taken place.

Figure 67 (A, B and C) is an example of a case of auricular flutter, in which tracings taken on successive days show, first, an alteration in the rate and rhythm of the ventricular responses and, finally, the appearance of complicating ventricular extrasystoles. Tracing A shows that the auricles were in a state of flutter and that the ventricular rate was moderate and perfectly regular. Because of dyspnea and a slight pretibial edema, digitalis was prescribed in moderate doses for a short period (in the course of three days 9½ grains were given). Tracing B, taken on the second day, showed that the ventricular rate was definitely slowed and became irregular. On the fourth

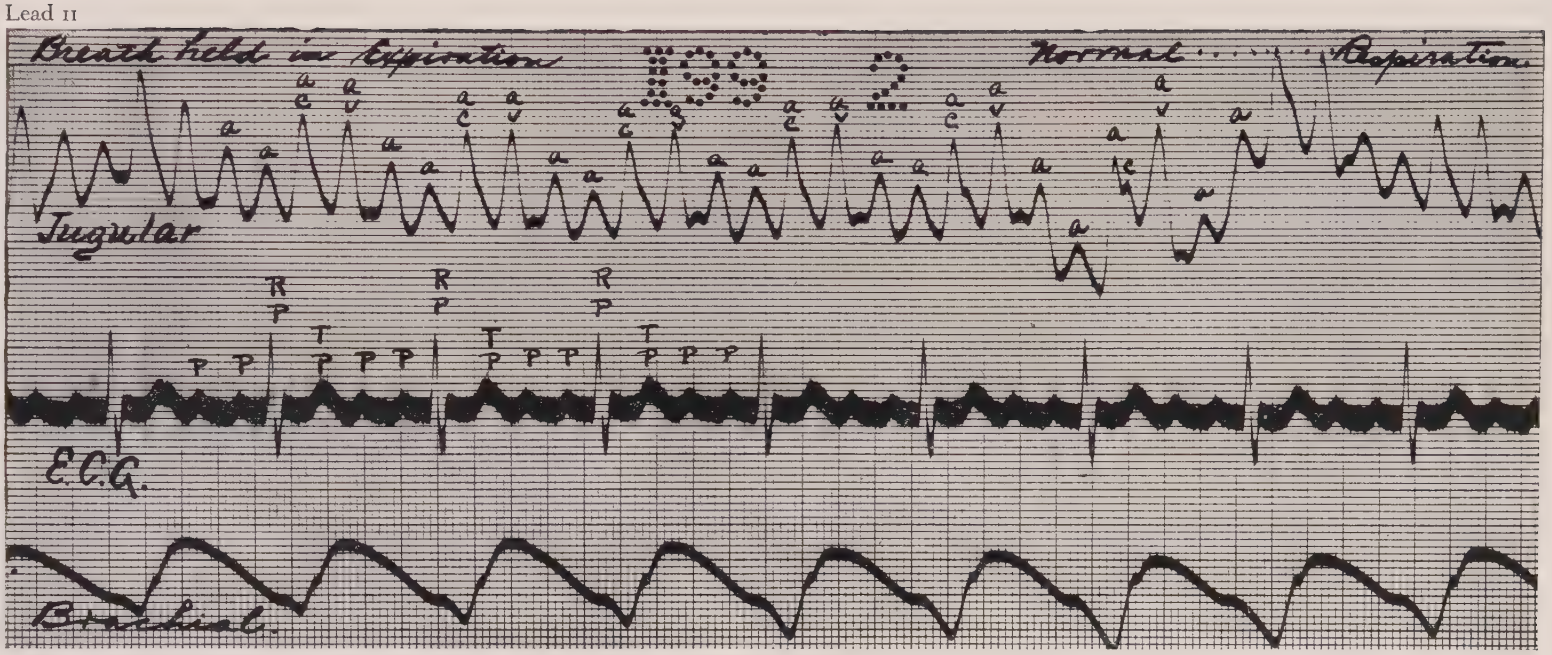


FIG. 67A.—The jugular tracing and the auricular P waves in the electrocardiogram show that the auricles are in a state of flutter. The ventricular complexes in the electrocardiogram (R-T) and the arterial pulse (brachial) show that the ventricular rate is moderate and perfectly regular.

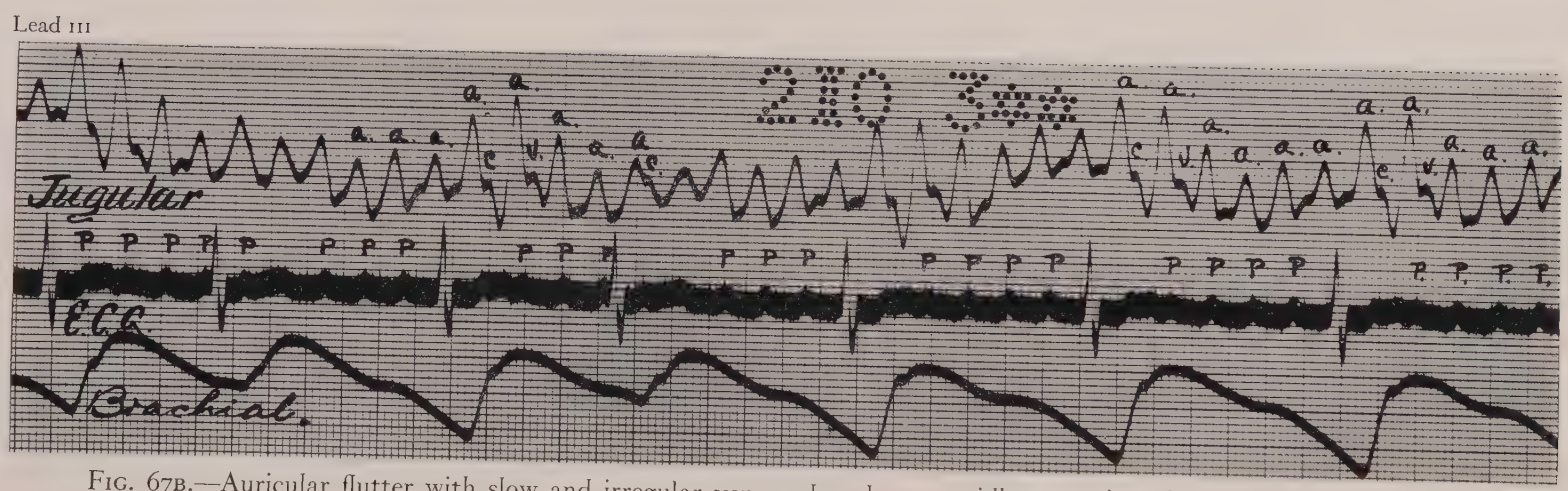


FIG. 67B.—Auricular flutter with slow and irregular ventricular response. The auricular P waves are shallow but equally spaced and regular. The synchronous jugular tracing also shows rapidly recurring rhythmic auricular A waves. (This tracing was taken two days later than tracing A.)

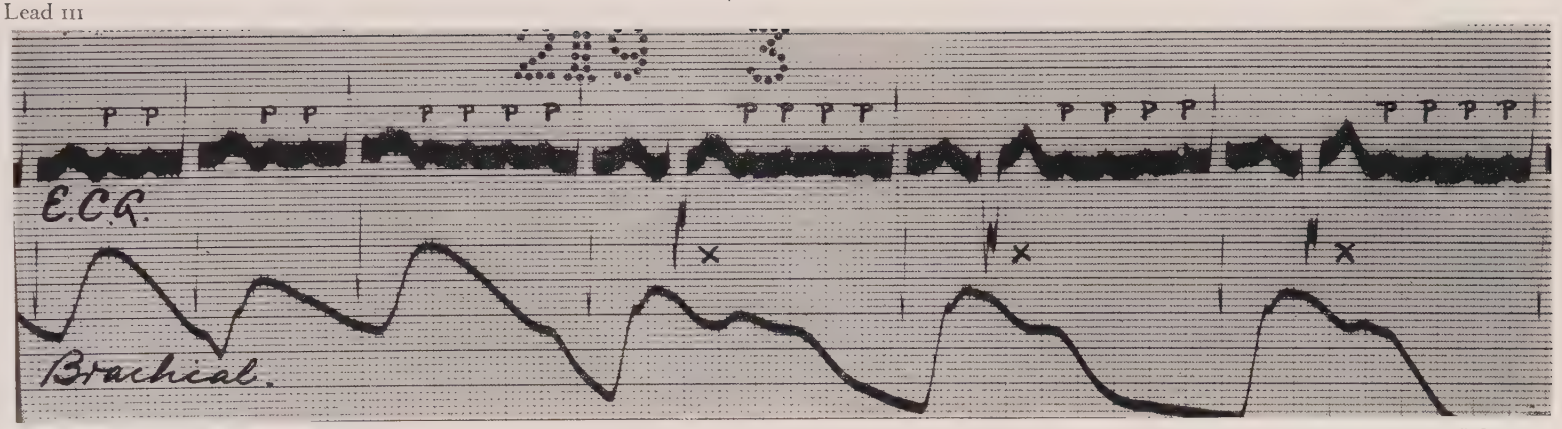


FIG. 67C.—Auricular flutter and ventricular extrasystoles. The rapidly recurring, rhythmic auricular P waves indicate the fundamental disturbance of mechanism as being that of auricular flutter. The ventricular responses are slow and irregular. Bizarre ventricular complexes, ventricular premature beats (x) are seen following cycles 4, 5 and 6. The ventricular extrasystoles are seen to induce feeble, premature pulse beats in the arterial pulse. (This tracing was taken four days later than tracing A.)

day, the extrasystoles appeared (Tracing C) and gave the patient a great deal of annoyance.[1]

Auriculo-ventricular Heart Block with Ventricular Extrasystoles (Fig. 68)

Auriculo-ventricular heart block is rarely accompanied by ventricular extrasystoles. As in the combined arrhythmias previously mentioned, it may be digitalis or one of its allied drugs that brings about the disorder. Both the heart block and the extrasystoles may be due to the same drug, especially if given in massive doses. When caused by the vagotropic effect of digitalis, its pharmacological antagonist, atropine, generally removes its effect at least temporarily.

Figure 69 (A, B, C and D) is presented to show the effect of atropine on such a combined disorder when induced by digitalis.

The first tracing (A) shows a complete auriculo-ventricular heart block with an occasional ventricular extrasytole as a result of excessive digitalis administration in the case of a patient with chronic valvular disease, who was brought into the hospital suffering from severe heart failure. Massive digitalization was employed. Approximately 300 minims of the tincture had been given within forty-eight hours. The amount of digitalis, if any, given before the onset of the attack could not be determined. The combined arrhythmia shown in the tracing developed promptly.

As a test to determine whether the disorder was caused by digitalis, the patient was given atropine sulphate gr. $\frac{1}{75}$ hypodermically. Electrocardiograms B, C and D were then

[1] Two days later, while attending light household duties "she suddenly felt very bad, became very short of breath and died within about one hour." The immediate ante-mortem cardiac disorder (possibly ventricular tachycardia or embolism) was not observed because death occurred before a physician could be summoned.

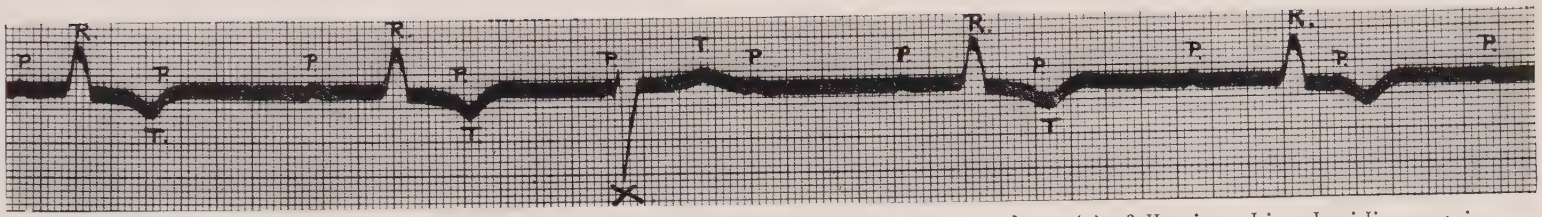


FIG. 68.—Complete auriculo-ventricular heart block with ventricular extrasystoles. The slow rhythmic idio ventricular rhythm (R — R) is interrupted in the 3rd cycle by a ventricular premature beat (x); following this, the idio-ventricular rhythm centre again dominates the ventricular rate.

FIG. 69.—The effect of atropine on auriculo-ventricular heart block.

A. Complete dissociation of auricles and ventricles. Auricular rate = 88.23 per minute. Ventricular rate = 60 per minute. There is an occasional ventricular extrasystole recorded.

B. Twenty minutes after a hypodermic injection of atropine sulphate, grains $\frac{1}{75}$. The rhythm is regular and each auricular P wave is followed by a ventricular complex. The P-R intervals are still definitely prolonged (0.28–0.32 sec.).

C. Fifty minutes after the atropine administration. The rhythm is perfectly regular, the P-R intervals are but slightly prolonged (0.24 sec.).

D. One hour and twenty minutes after atropine administration. In the first five cycles the P-R intervals are seen to be progressively prolonged. Following this the heart block suddenly reappears, associated with ventricular extrasystoles.

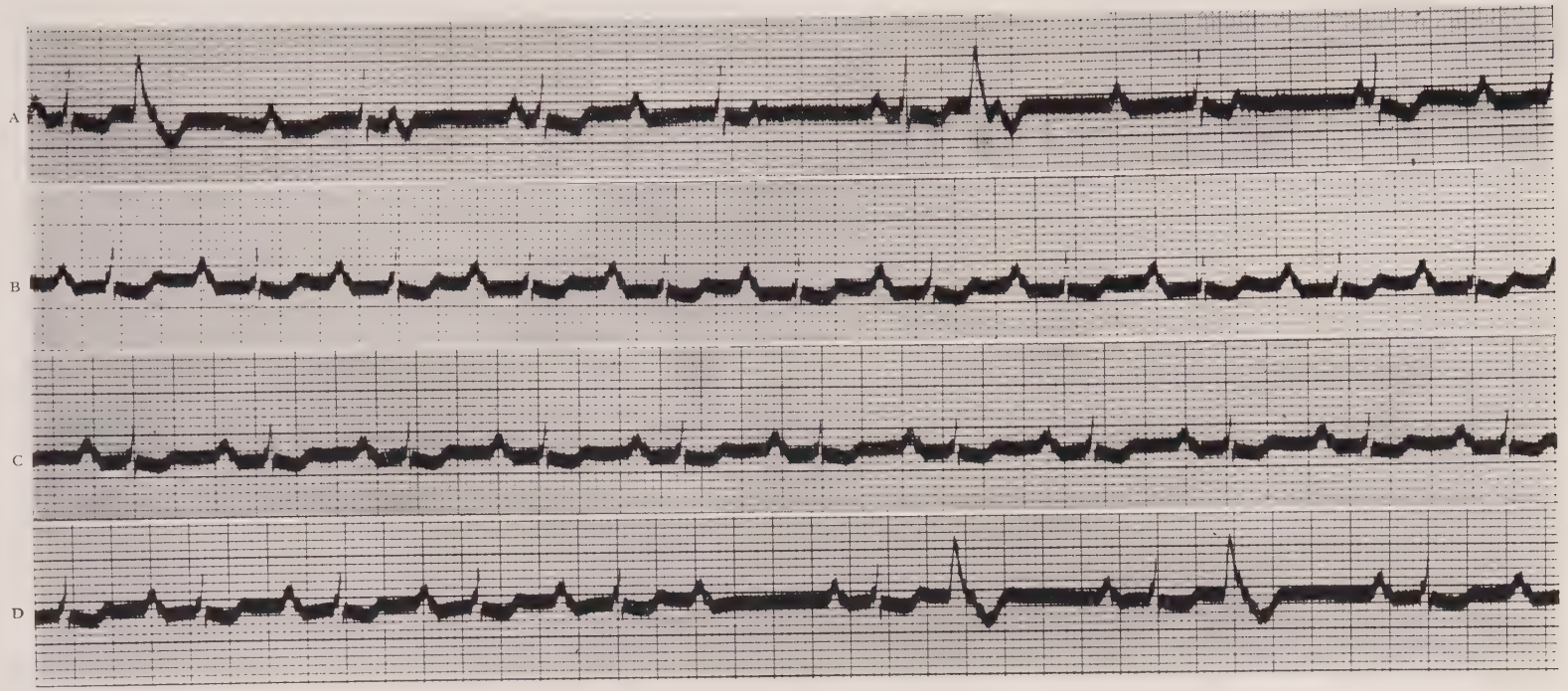


Fig. 69.

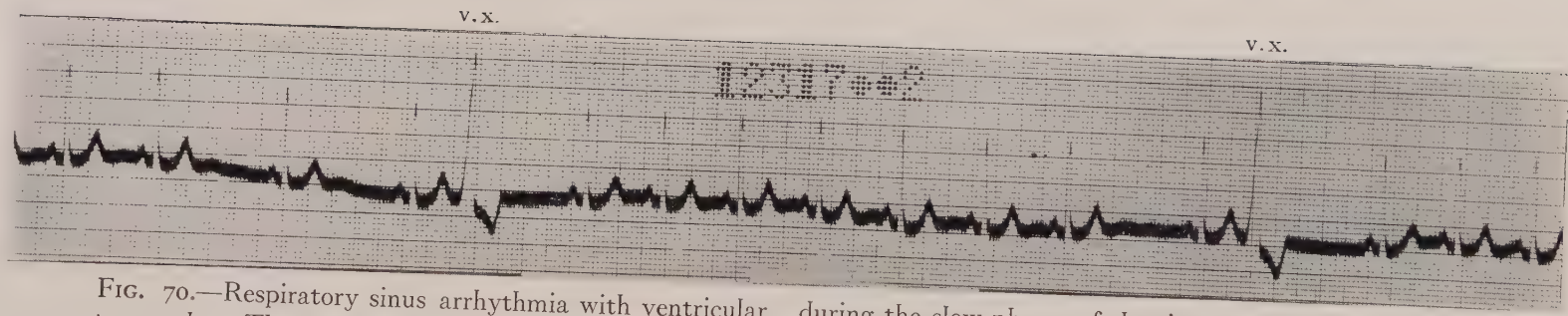


Fig. 70.—Respiratory sinus arrhythmia with ventricular extrasystoles. The ventricular extrasystoles (v.x.) appear during the slow phases of the sinus arrhythmia.

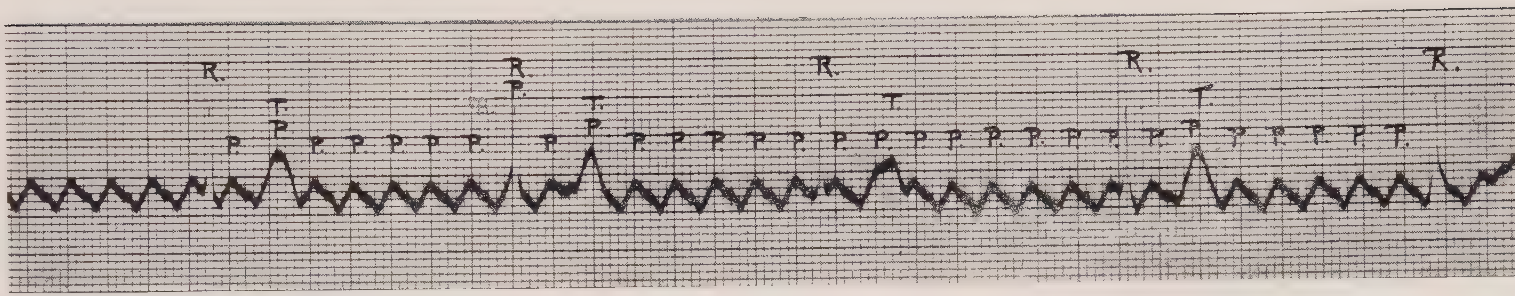


Fig. 71.—Auricular flutter and complete A-V heart block. The auricles are in a state of flutter as shown by the rhythmic auricular P waves recurring at a rate of approximately 300 per minute. The ventricles apparently respond to an "idioventricular" rhythm at a rate of approximately 40 per minute (R and T waves).

taken. Tracing B was taken twenty minutes after the administration of the atropine. It shows that the rhythm had become regular but that there was still considerable delay in A-V conduction, in some cycles as high as 0.36 second. The third tracing (C) was taken about fifty minutes after the atropine administration. It shows a normal rhythm and a moderately rapid rate. The P-R interval in this tracing is practically within the upper limit of normal auriculo-ventricular conduction time. Tracing D was taken one hour and a half after the administration of atropine. It shows the return of the heart block and the reappearance of the extrasystoles.

Sinus Arrhythmias Combined with Other Disorders

Respiratory sinus arrhythmia, being a very frequent disorder, is often accompanied by other irregularities. Any of the other sinus arrhythmias may be associated with it. At times, premature beats, either auricular or ventricular, may complicate it. Figure 70 shows a respiratory sinus arrhythmia accompanied by ventricular premature beats.

Superimposition of Disorders Inducing Regularity of Heart Beat

As has been mentioned, the superimposition of one disorder upon another may render a previously irregular heart beat regular. Figure 65 shows an auricular fibrillation with complete auriculo-ventricular heart block. The slow and regular ventricular rhythm is motivated by an independent "Idio-ventricular Center" of impulse production.

Similarly in Figure (71) where the auricles are in a state of flutter, the ventricles are slow and regular because of an associated complete auriculo-ventricular heart block.

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